TRANSACTIONS

AMERICAN ACADEMY

of

OPHTHALMOLOGY

and

OTOLARYNGOLOGY

SEPTEMBER - OCTOBER
1958

Volume 62 No. 5

ANNUAL MEETING, OCTOBER 11-16, 1959, CHICAGO

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American Academy of Ophthalmology and Otolaryngology

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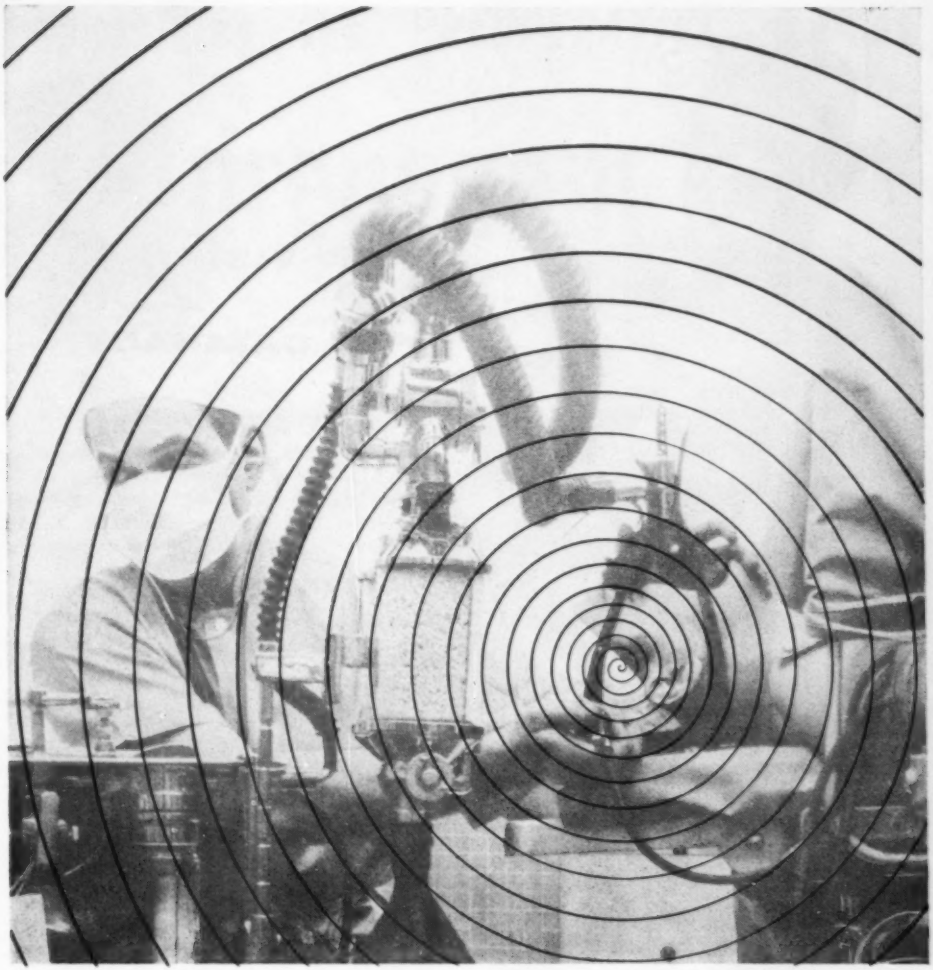

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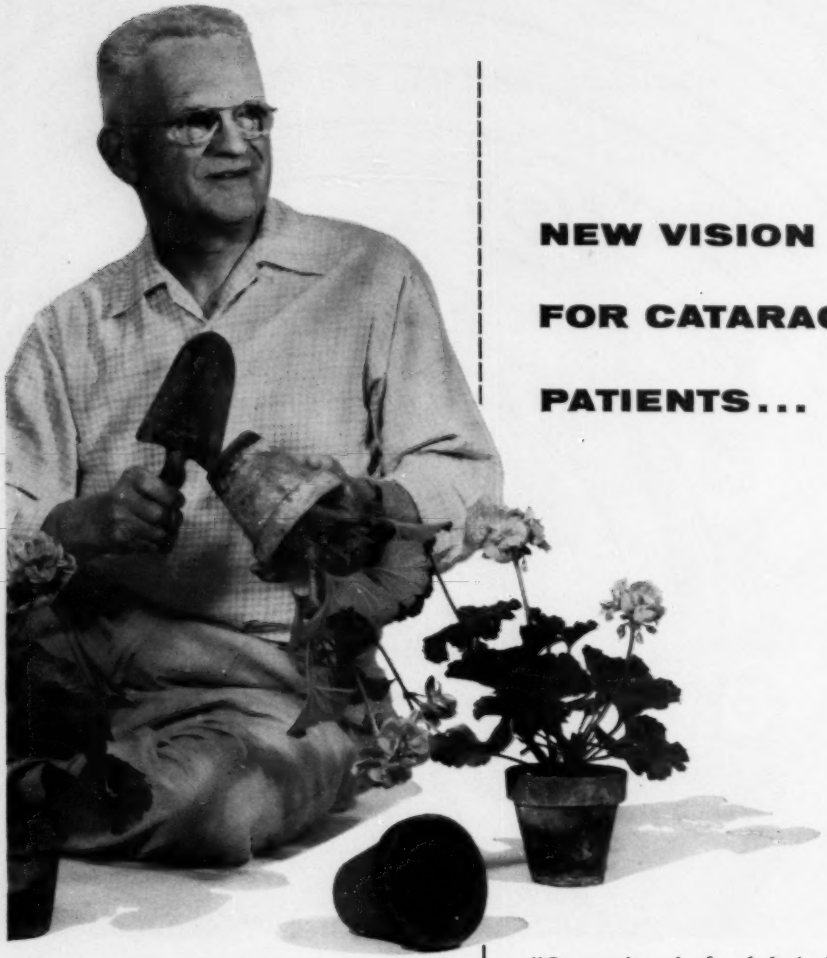

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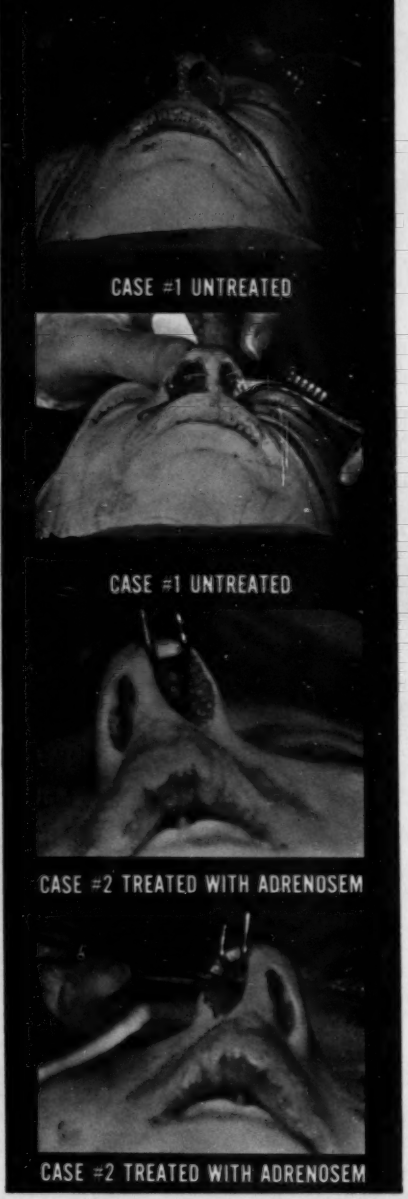

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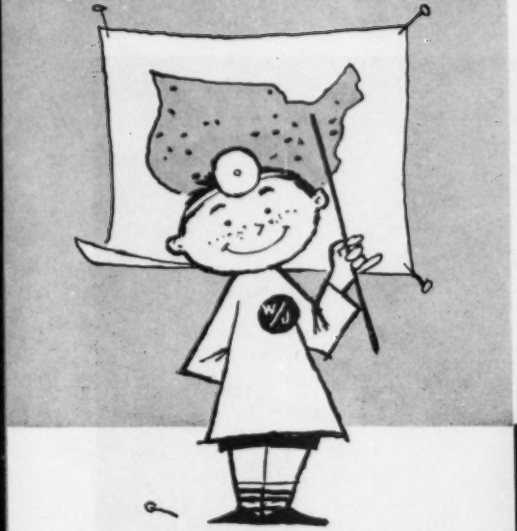

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Los Angeles	Wed. Oct. 29	7 to 10 P.M.	A & B	Statler
Oakland	Fri. Oct. 24	7 to 10 P.M.	A	Leamington
San Diego	Thur. Oct. 30	7 to 10 P.M.	A	El Cortez
San Francisco	Sun. Oct. 26	1 to 5 P.M.	A & B	St. Francis
COLORADO				
Denver	Tues. Nov. 4	7 to 10 P.M.	A & B	Brown Palace
DISTRICT OF COLUMBIA				
Washington	Sun. Nov. 23	1 to 5 P.M.	A & B	Statler
FLORIDA				
Jacksonville	Tues. Dec. 16	7 to 10 P.M.	A	Roosevelt
Miami	Sun. Dec. 14	1 to 5 P.M.	A	McAllister
Tampa	Sun. Dec. 14	1 to 5 P.M.	A	Floridan
GEORGIA				
Atlanta	Thur. Dec. 11	7 to 10 P.M.	A & B	Atlanta Biltmore
Savannah	Tues. Dec. 16	7 to 10 P.M.	A	De Soto
KENTUCKY				
Louisville	Thur. Dec. 18	7 to 10 P.M.	A	Brown
MARYLAND				
Baltimore	Fri. Nov. 21	7 to 10 P.M.	A	Lord Baltimore
MASSACHUSETTS				
Boston	Sun. Nov. 16	1 to 5 P.M.	A & B	Statler
NEW YORK				
Buffalo	Thur. Nov. 13	7 to 10 P.M.	A & B	Statler
New York City	Tues. Nov. 18	7 to 10 P.M.	A & B	The Savoy Hilton
OREGON				
Portland	Fri. Oct. 24	7 to 10 P.M.	A	Multnomah
PENNSYLVANIA				
Philadelphia	Thur. Nov. 20	7 to 10 P.M.	A & B	Sheraton
TENNESSEE				
Nashville	Thur. Dec. 18	7 to 10 P.M.	A	Dinkler-Andrew Jackson
UTAH				
Salt Lake City	Sun. Nov. 2	1 to 5 P.M.	A & B	Utah
VIRGINIA				
Norfolk	Tues. Dec. 9	7 to 10 P.M.	A	Commodore Maury
Richmond	Tues. Dec. 9	7 to 10 P.M.	A	John Marshall
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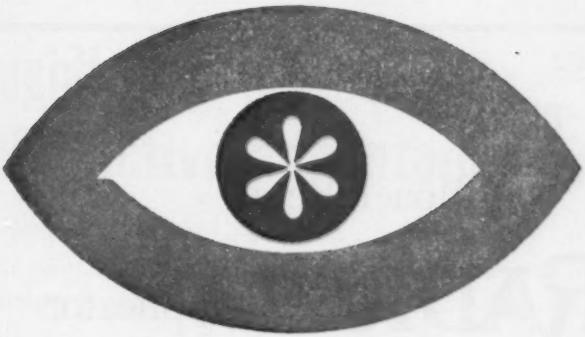

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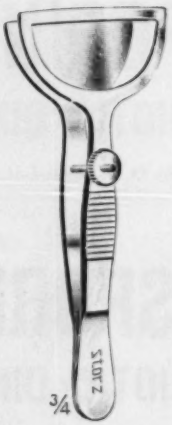

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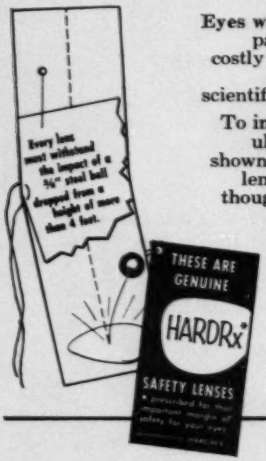
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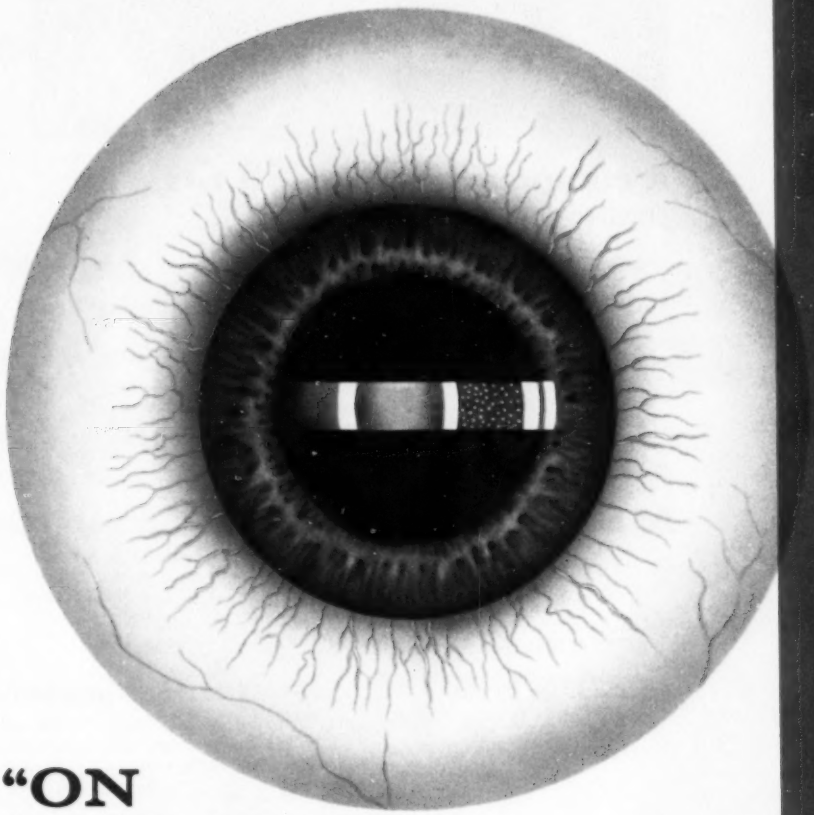

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It is a direct-reading, one-position instrument which gives you an objective method for quickly determining axis and power of corneal astigmatism. In addition, its easily manipulated simple settings measure the radius of curvature of the cornea to aid in determining proper curvature of the contact lens. These measurements are not easily accomplished by other means.

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You can adjust the compact AO Micromatic Ophthalmometer to your patient precisely and quickly. You'll find all readings and controls in comfortable, natural positions; easy to adjust, easy to read.

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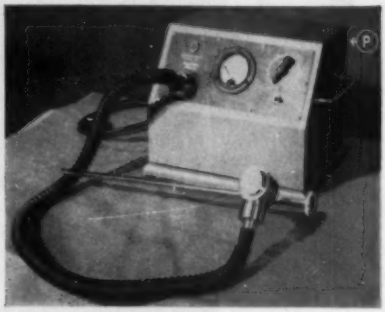
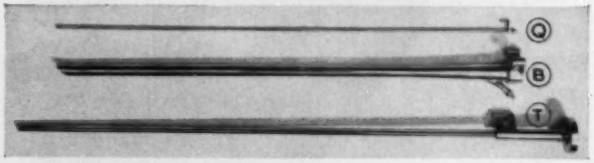
The Chevalier L. Jackson Fotobronchoscopic Unit for endobronchial photography

still or motion pictures / color or black and white

For diagnostic study, record keeping, clinical presentation and instruction...this set of instruments is the complete accessory unit needed for fine clinical picture-taking with your own camera.



bronchoscope 9mm x 40cm (B)
quartz rod for illumination (Q)
forward-viewing telescope (T)
luminator (L)
power unit (P)



Optics, certified by the Optical Institute, are imported from France.

The bronchoscope is of special design with light carrier canal inside the tube. This canal accommodates the standard Jackson light carrier and lamp, as well as the new quartz rod—thus the bronchoscope may be used in a routine fashion for regular procedures, or, with a quartz rod for photography.

The quartz rod is specially treated to transmit maximum light from the luminator to the distal end of the tube. These rods are encased in BRONCHALLOY, giving them a strength which belies the delicate design.

The forward-viewing telescope is complete with an anti-fogging device: an air jet across the face of the distal telescope lens assures constant clear vision. Adaptor rings are available to make proper contact between the proximal telescope eye-piece and the camera lens.

The luminator is the heart of the instrument . . . engineered so that its use is simple but very effective. The lamp in the luminator produces excellent light transmitted distally by the quartz rod. It is air-cooled by a large capacity pump housed in the power unit. Rheostat controls on the power unit provide variable light—four volts for regular bronchoscopy, up to 10½ volts for "over-illumination" of the lamp for the white light ideal for picture taking.

References:

Jackson, C.L.: New Instrumentarium for Peroral Endoscopy and Endoscopic Photography. LARYNGOSCOPE, *67*:777, (Aug.) 1957.

Fourestier, M., Gladu, A., and Vulmiere, J.: LA PRESSE MEDICALE, *60*:1292, (Oct.) 1952.

Dubois De Montreynaud, J.M., Edwards, Roger, J., and Gladu, Amédé J.: LARYNGOSCOPE, *66*:637, (June) 1956.

——JOUR. BIOLOG. PHOTOG. ASSOC., *24*:45, (May) 1956.

The Jackson Fotobronchoscope is on display in Pilling's Philadelphia store and New York office. Write Philadelphia for further information and prices; send orders to Philadelphia.

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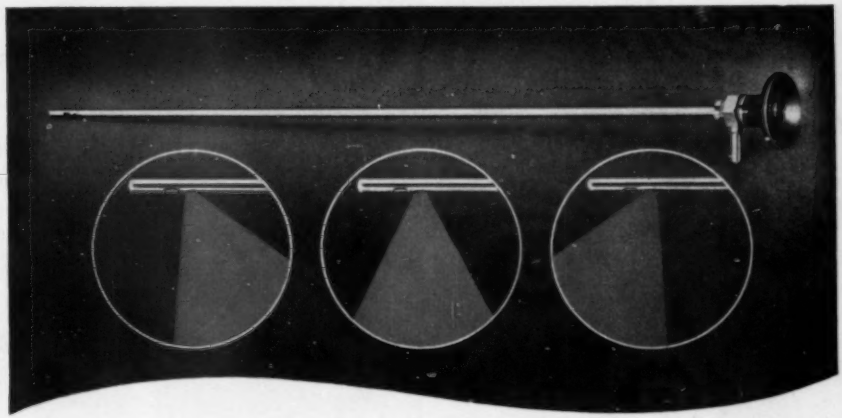

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Perfect compatibility with Jackson bronchoscopes and equipment

Pilling-made telescopes offer the bronchoscopist the advantages of working with a perfectly balanced instrument with superior optical system and trouble-free electrical design.

The basic design assures easy service and replacement of parts.

OPTICAL FEATURES: The lens system gives maximum color correction and clarity with minimum distortion. Three styles—

Forward-Oblique,	approximately 55° field of vision, 48 cm. long
Br 1400	5.4 mm. diameter for use with 6 mm. full lumen or 7 mm. standard bronchoscope
Retrospective,	approximately 55° field of vision, 48 cm. long
Br 1405	4.8 mm. diameter for use with 5 mm. full lumen or 6 mm. standard bronchoscope
Br 1406	5.4 mm. diameter for use with 6 mm. full lumen or 7 mm. standard bronchoscope
Right Angle,	approximately 55° field of vision, 48 cm. long
Br 1410	4.8 mm. diameter for use with 5 mm. full lumen or 6 mm. standard bronchoscope
Br 1411	5.4 mm. diameter for use with 6 mm. full lumen or 7 mm. standard bronchoscope

ELECTRICAL FEATURES: The lamp is designed especially for long life. Made with a hard rubber insulated and soldered contact, it has a positive electrical connection at all points and at all times, and avoids two sources of trouble—wires and pigtail contacts.

A bayonette connection identical with all Jackson bronchoscopes makes it possible to use the same cord for the telescope current as for the bronchoscope—one type cord in use throughout all bronchoscopic procedures.

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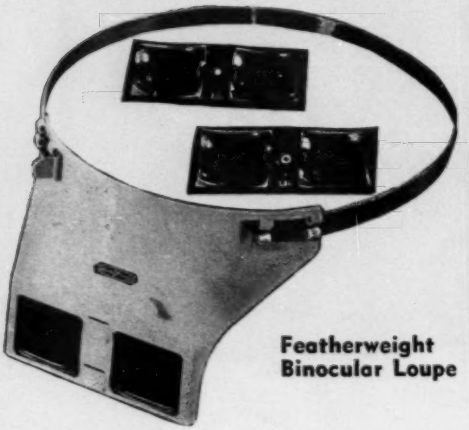

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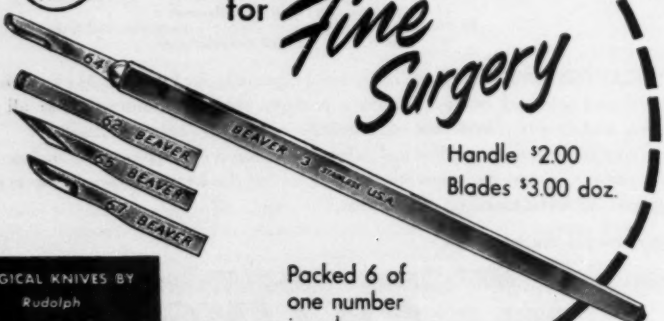

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Dichlorphenamide

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- minimal loss of effectiveness, even with long-term use
- smooth control—few side effects
- low dose effectiveness—less than with other carbonic anhydrase inhibitors
- may be effective when other therapy, including miotics, has failed or has not been tolerated
- effective orally—unsurpassed absorption after oral administration

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'DARANIDE'
Tablets

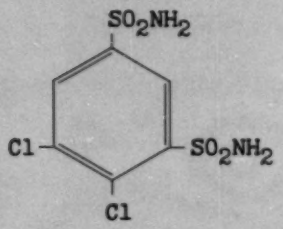
Dichlorphenamide

DESCRIPTION

'Daranide' is a new, orally effective, potent carbonic anhydrase inhibitor. Because of its marked inhibitory effect on this enzyme in the eye, 'Daranide' is a therapeutic agent of considerable value in reducing the increased intraocular pressure in many types of glaucoma.

CHEMISTRY

'Daranide', a dichlorinated benzenedisulfonamide, is known chemically as 1,3-disulfamyl-4,5-dichlorobenzene and is designated by the generic name of dichlorphenamide. It has the following structure:



MODE OF ACTION

'Daranide' is primarily a carbonic anhydrase inhibitor; i.e., it interferes with the catalytic activity of this enzyme, which is concerned with the formation of H^+ and HCO_3^- ions and the conservation of base as sodium bicarbonate. Under the influence of 'Daranide' the renal excretion of sodium, potassium, and bicarbonate is increased and an alkaline urine is produced. However, this compound also has the

advantage of causing a definite increase in chloride excretion. Partially as a result of the greater excretion of chloride, 'Daranide' does not produce metabolic acidosis as readily as other compounds of this nature. Furthermore, in animal studies 'Daranide' has been shown to maintain its essential activity over the course of weeks or months.

The compound does not appear to be concentrated in gastric mucosa in sufficient amounts to influence materially the activity of carbonic anhydrase in this tissue. Thus, in dogs, gastric motility or secretion is not affected to any significant degree by the administration of 'Daranide'.

PHARMACOLOGY

'Daranide' is uniformly and well absorbed following its oral administration; it is essentially as active when given orally as parenterally. The enhancement of sodium, potassium, and chloride excretion by the oral administration of a single dose is rapidly manifested and persists for a number of hours, indicating a prolonged duration of action of the compound. The urinary pH is elevated as the result of sustained bicarbonate excretion. However, greater chloruresis and less acidosis are induced than with other carbonic anhydrase inhibitors, with consequent greater efficacy on repeated administration. The natriuretic effect of 'Daranide' has been observed even after 10 weeks of continued administration to dogs. The activity of 'Daranide' is not impaired in the presence of an acid urine such as that accompanying the administration of ammonium chloride.

'Daranide' has a low inherent toxicity when administered orally or parenterally to laboratory animals. The compound has been given orally to dogs and rats over a period of at

least six months with essentially no evidence of toxicity. Pharmacologic investigation indicates an absence of irritating effect on the mucosa of the eye as demonstrated by a lack of local anesthetic activity or irritation when a 1 per cent concentration of the drug (suspended in tragacanth) is instilled into the conjunctival sac of the rabbit eye.

CLINICAL EVALUATION

Effect on Intraocular Tension

Studies in humans have demonstrated that 'Daranide' administered orally reduces intraocular pressure in both glaucomatous and nonglaucomatous eyes. The onset of action following a single dose is within an hour and maximal effect is observed within two to four hours The lowered intraocular tension is maintained for approximately six hours. It has been possible to control excessive intraocular pressure in patients with glaucoma for a period of many months by the daily combined use of 'Daranide' and miotics. Tonographic studies have shown that 'Daranide' lowers intraocular pressure by inhibiting the formation of aqueous humor in normal as well as in glaucomatous eyes.

Effect on Electrolytes

In the human as in the laboratory animal, 'Daranide' enhances the excretion of sodium, potassium, bicarbonate, and water. An increased chloride excretion greater than that following the use of other carbonic anhydrase inhibitors is also observed with 'Daranide'. This enhanced excretion of chloride explains, at least in part, the continued efficacy of 'Daranide' with repeated administration and the lesser propensity for the development of metabolic acidosis.

As is the case with all carbonic anhydrase inhibitors, 'Daranide' in high doses causes some

decrease in renal blood flow and glomerular filtration rate.

INDICATIONS

'Daranide' is indicated for oral treatment of various types of glaucoma:

chronic simple (wide angle) glaucoma
acute congestive (narrow angle) glaucoma
chronic congestive glaucoma
secondary glaucoma (acute phase)
preoperative control of intraocular tension of glaucoma

DOSAGE

The usual adult dosage range is from 25 to 50 mg. (½ to 1 tablet) once to three or four times daily. Dosage must be carefully adjusted to meet the requirements of the individual patient.

Therapy with 'Daranide' is usually most successful when employed in conjunction with miotics such as pilocarpine, physostigmine, 'Floropryl' (isoflurophate), or 'Carcholin' (carbachol). In acute congestive glaucoma, 'Daranide,' with miotics, should be administered promptly in an attempt to bring about rapid reduction of intraocular tension. If this is not quickly relieved, surgery may be mandatory. In chronic congestive glaucoma, surgery may be necessary if the intraocular tension is not brought successfully under control after an adequate trial period of therapy with 'Daranide'.

PRECAUTIONS

Since 'Daranide' is a carbonic anhydrase inhibitor, certain side effects characteristic of this class of compounds will occur, particularly in the higher dosage ranges. These effects may in-

clude anorexia, nausea, vomiting, nervousness, lassitude, depression, confusion, disorientation, sedation, dizziness, ataxia, tremor, tinnitus, paresthesias, and pruritus. Fortunately, the therapeutic amount of 'Daranide' required for most patients is well beneath the level that will produce these effects. When they do occur, reduction of dosage or temporary discontinuance of the drug is indicated.

It is essential to keep a close check on the electrolyte balance of the patient during therapy with 'Daranide', as with patients on any other carbonic anhydrase inhibitor.

SUPPLIED

'Daranide' Tablets, 50 mg., bottles of 100 and 1000.

REFERENCES

Becker, B.: Glaucoma, 1956-1957, A.M.A. Arch. Ophth. 58: 862, Dec. 1957.

Crosley, A.P., Jr., and Cullen, R.E.: Effects of Dichlorphenamide on Renal Hemodynamics and Electrolyte Excretion, Fed. Proc. 16: 289, March 1957.

Gonzales-Jimenez, E., and Leopold, I.H.: The Effect of Dichlorphenamide (A Carbonic Anhydrase Inhibitor) on the Intraocular Pressure of Humans, A.M.A. Arch. Ophth. (in press).

Harris, J. E., Beaudreau, O., and Hoskinson, G.: Clinical and Laboratory Experiences with the Carbonic Anhydrase Inhibitor, Dichlorphenamide, Am. J. Ophth. 45: 120, Jan. 1958.

'Daranide', 'Floropryl' and 'Carcholin' are trademarks of Merck & Co., Inc.

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Aside from the cases of hearing loss that can be relieved by medical treatment, there are millions who suffer from partial impairment that can be helped by a hearing aid. Yet, today an estimated *80% of the hard-of-hearing whose condition could be improved electronically are not wearing a hearing aid.*

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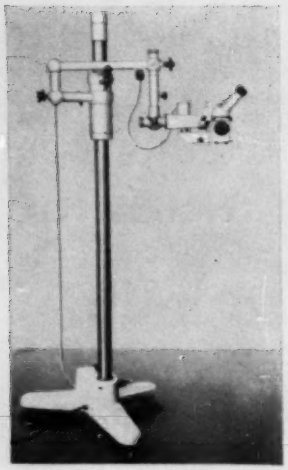

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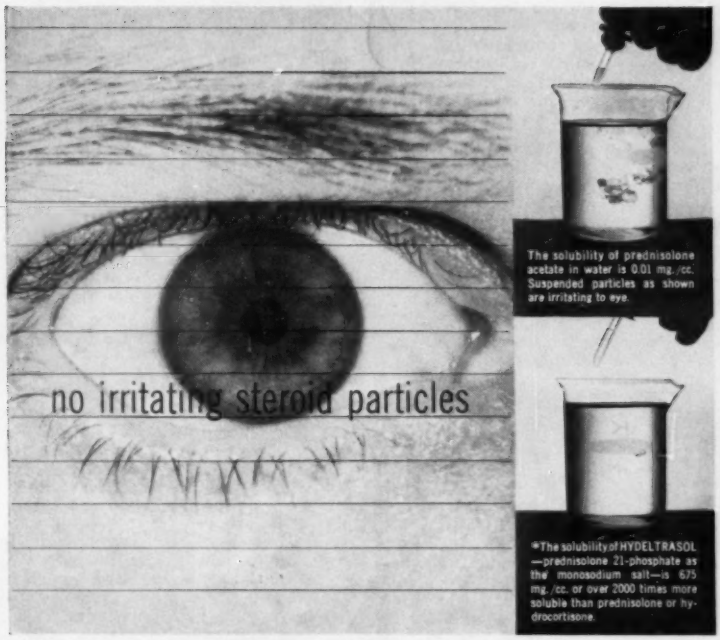
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Telegraph collect if specimen is being sent. Send eyes to . . .

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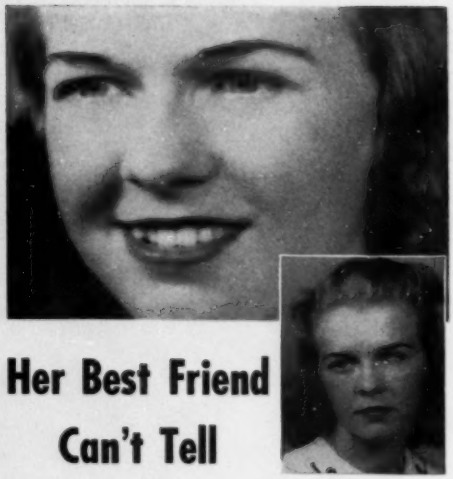

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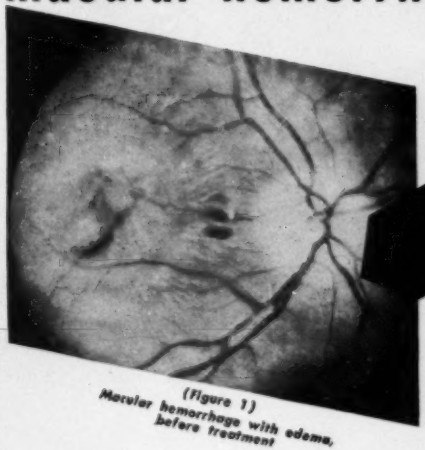
macular hemorrhages

respond to IODO-NIACIN*



(Figure 1)
Macular hemorrhage with edema, before treatment

(Figure 2)
After 25 days' treatment with Iodo-Niacin

Macular and retinal hemorrhages are satisfactorily absorbed after use of IODO-NIACIN[1]. The dosage used in this study was 1 tablet three times daily.

In cases of cerebral and generalized arteriosclerosis, IODO-NIACIN has been used with great benefit[2]. Full doses were given for a year or longer without any iodism or ill effects.

The formula of IODO-NIACIN Tablets contains potassium iodide 135 mg. (2¼ gr.) combined with niacinamide hydroiodide 25 mg. (⅜ gr.). IODO-NIACIN Ampuls 5 cc. are highly satisfactory for intramuscular or intravenous use[3].

Since the retina is intrinsically a part of the brain[4], hemorrhages in this region are indicative of a similar cerebral condition for which IODO-NIACIN is directly indicated.

IODO-NIACIN Tablets, slosol coated pink, are supplied in bottles of 100. Ampuls 5 cc. in boxes of 10.

1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.
4. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1598.

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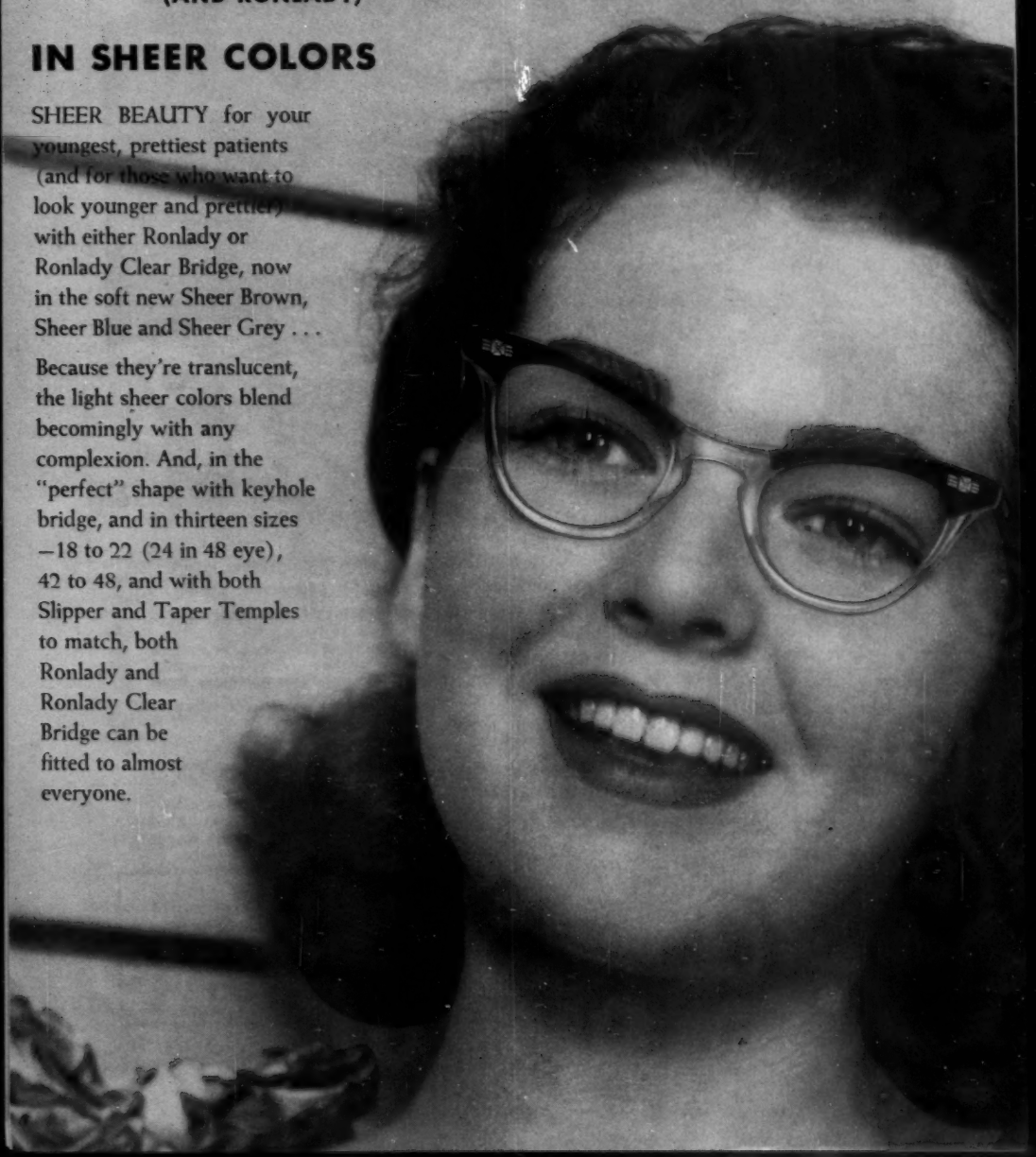

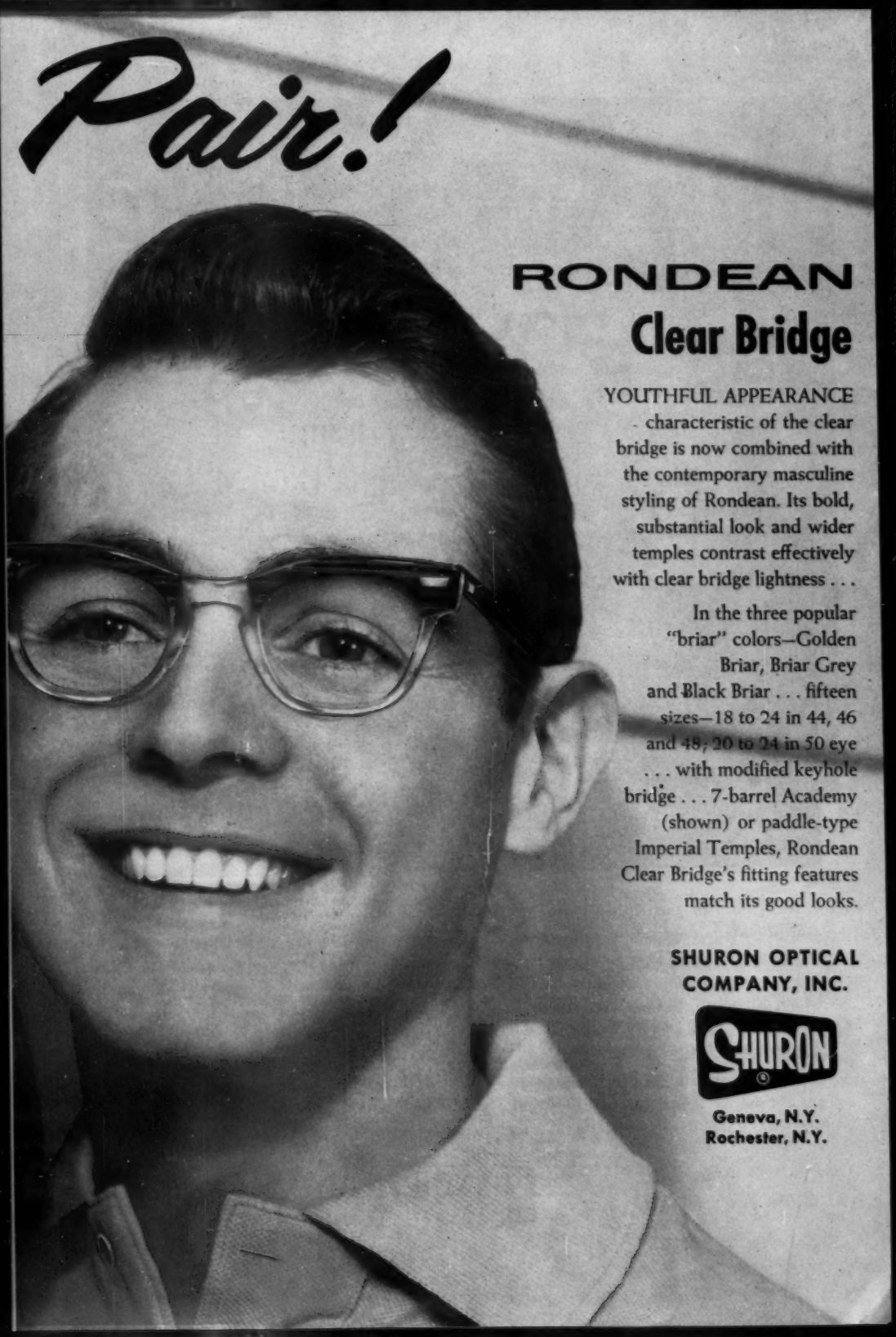
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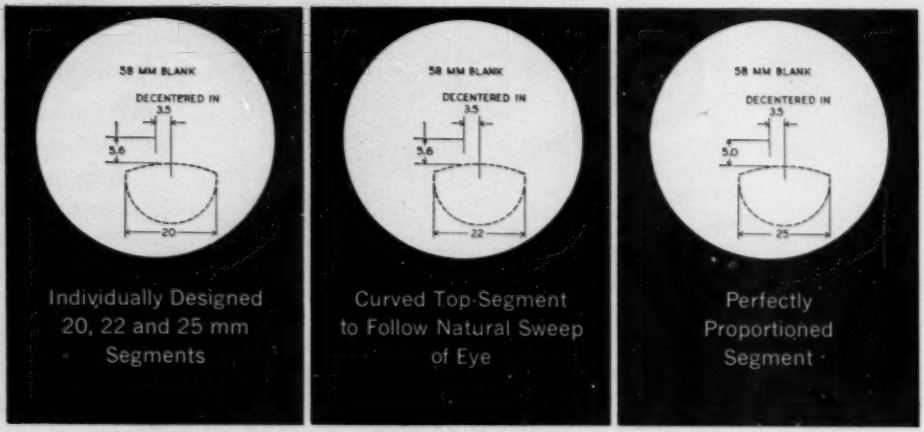

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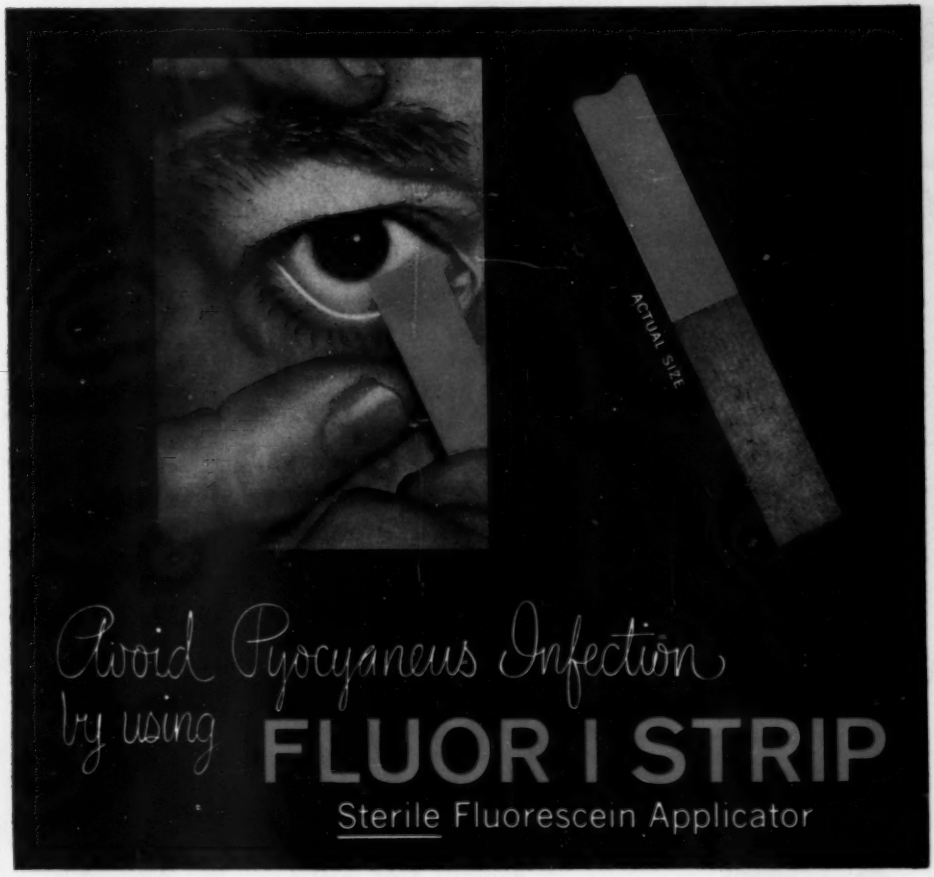

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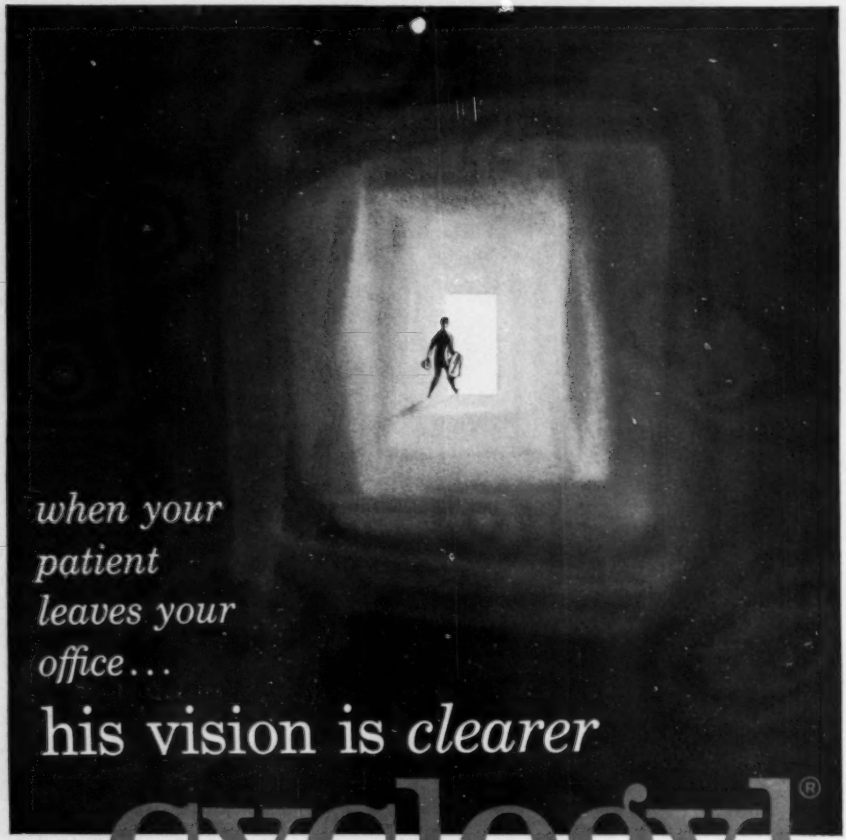

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My role in this symposium is to review briefly the anatomy of the facial skeleton. In doing so I shall endeavor to emphasize the fact that the bony components of the face are so intimately related that injury to one may easily result in injury to a number of others, involving not only the bones of the face but also those of the cranium.

The skull as a whole is a union of individual bones. Some of these form a skeletal container for the brain and are commonly referred to as the bones of the cranium; these include the paired parietal and temporal bones, and the unpaired ethmoid, frontal, occipital and sphenoid bones. Others form the facial skeleton; these include the unpaired mandible and vomer, and the paired lacrimal, nasal, inferior turbinate, maxillary, zygomatic and palatine bones. Joined by various irregular articulating sutures, the bones of the face form a homogenous framework which acts as a support and a protective base for the softer structures.

In addition to their contribution to the bony envelope of the brain, a number of the cranial units also play a role in supporting various portions of the facial skeleton; these are the ethmoid, frontal, sphenoid and paired temporal bones. Because of this anatomic kinship, one should retain an awareness of the fact that some of the bones of the skull, although described as bones of the cranium, are not limited to that category alone.

The strong rim of the orbit is formed above by the frontal bone; the zygomatic bone constitutes its lateral margin; the zygoma and the maxilla both contribute to the contour of the lower rim, and the maxilla and frontal bones together form the median segment of the anterior orbital framework. The medial margin turns in toward the lacrimal groove; the other borders are readily palpable through the overlying soft tissues. The external angular process of the frontal bone is situated at the superior lateral margin of the orbit; the suture which it forms with the orbital process of the zygoma is palpable from outside the face both on its orbital and temporal aspects.

The contour of the orbit may be distorted by comminution of bone and telescoping of bony fragments in maxillary and zygomatic fractures. Separation of the frontozygomatic junction may occur in fractures of the zygomatic bone. In such a condition, the lateral canthus and the orbital contents are usually displaced downward, the lower eyelid is retracted and a deepening of the superior palpebral sulcus results from ptosis of the ocular globe.

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The plate of bone which separates the roof of the maxillary sinus from the orbital floor is thin and subject to fracture, thus involving the orbital contents and the antrum; the frontal sinus and ethmoidal air cells are also in close proximity to the orbit.

The nasal bones together form the bridge of the nose; they are attached to one another in the midline, to the nasal notch of frontal bone above, to the lateral cartilage of the nose below, and laterally to the frontal process of the maxilla. A bony crest in the midline of the under surface of each of the nasal bones articulates with the ethmoidal contribution to the bony nasal septum. The upper portion of the nasal bone is thicker and stronger than the lower portion, resisting fracture; the lower portion is more often fractured. Such fractures usually are compound because of the thinner and more intimate relation of the mucosa to the osseous structure.

Because of the anatomic relations of the nasal bones to the perpendicular plate, and the crista galli of the ethmoid bone to the olfactory bulbs in the anterior cranial fossa, severe injury to the bones of the nose may result in destruction of the olfactory sense or in septic meningitis; in seeking a cause for such conditions, a fracture of the nasal bones is often found. The nasolacrimal duct may be occluded in nasal or maxillary fractures.

Injuries of the lacrimal bones are always compound fractures due to the close adherence of the mucosa, accounting in part for the profuse hemorrhage accompanying such trauma. When the cribriform plate of the ethmoid is fractured, the dura may be involved.

A view of the skull from below demonstrates the outward projection of the zygoma, strong abutment of the facial skeleton and most prominent feature of the cheek. The zygoma joins the zygomatic process of the maxilla anteriorly and the zygomatic process of the temporal bone posteriorly, completing the zygomatic arch which is thus formed of three bones: the zygoma, keystone of the arch, and the maxilla and temporal bones. The heavy converging fibers of the temporalis muscle insertion pass within the zygomatic arch to reach the coronoid process of the mandible. The inner surface of the zygoma is concave and faces the temporal and infratemporal fossae. The curved orbital surface faces the orbit externally and assists in the formation of the lateral wall and floor of the orbit.

Fractures of the zygoma are common because of its prominence on the face. Fracture of the body of the bone may involve the orbit, resulting in subconjunctival extravasation of blood in the subcutaneous tissues near the outer canthus. Because the zygomatic process is weaker toward its temporal portion it is broken more readily in that region; the fragments, when forced into the fibers of insertion of the temporalis muscle on the underlying coronoid process of the mandible, may be the cause of ensuing trismus or immobility of the mandible. The attachments of the strong temporalis fascia to the upper margin of the bone and the masseter muscle on the lower border usually prevent displacement of the fractured zygoma in a superior or inferior direction.

The maxilla, largest of the bones of the face, assists in the formation of the bony orbit, floor of the nose, zygomatic arch and roof of the oral cavity; it contains the large maxillary sinus, and is in intimate association with the frontal, nasal, lacrimal, ethmoid, inferior turbinate, vomer and palatine bones.

Fractures of the maxilla are usually caused by a forceful blow, direct impact being necessary to break the bone. The results of such trauma vary from simple localized separations to exceed-

ingly extensive injuries of the facial skeleton in which the nose, the sinuses, the orbits, the palate and the alveolar processes and teeth may suffer serious damage.

Unlike similar injuries to the lower jaw, the muscles attached to the anterior and lateral surfaces of the maxilla (muscles of facial expression) play an insignificant role in the displacement of separated fragments. Transverse fractures of the maxilla, however, may result in downward and backward displacement of the separated fragment because of the weight of the detached bone and the downward and backward traction exerted by the internal pterygoid muscles. In the reduction of such fractures, the maxilla must be drawn forward and upward; gentle rather than forceful pressure should be employed in these procedures in order to prevent such complications as the blocking of the nasolacrimal duct or the nasal passages.

The principal muscles that move the mandible are the masseter, temporalis and internal and external pterygoids. All the elevators of the jaw are inserted on the ramus of the mandible; thus a fracture anterior to the angle of the jaw results in an elevation of the separated ramus. When the neck of the mandibular condyle is fractured, the external pterygoid, which inserts into the condylar neck, exerts a medial pull upon the head of the condyle, resulting in an overriding of the fragment by the ramus due to the action of the elevator muscles of the jaw.

A blow from beneath the jaw in an upward and backward direction may result in fracture of the thin plate of bone which forms the mandibular fossa of the temporomandibular joint, and may also involve the tympanic component of the fossa.

When a fragment of the body of the mandible is lost due to severe fracture of the bone, the medial pull of the pterygoids on both sides of the head results in a contraction of the mandibular arch; treatment of such a fracture requires that the arch be spread to its normal width and bone transplanted to fill the gap.

In fractures of the lower jaw, displacement of the separated fragments is caused chiefly by two factors — the direction of the line of fracture and the muscle pull. It seems obvious that the result of the action of the muscles in the unbroken mandible is not identical with that in the fractured jaw, and that a multiplication of fractures in the same jaw complicates the degree of displacement.

THE TREATMENT OF MIDFACIAL FRACTURES

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FOR many years we employed the usually advocated procedures of blind reduction of facial fractures, especially those of the zygomatic compound. Furthermore, we relied upon the highly recommended usage of natural impaction or external fixation to maintain reduction. A few instances of less than ideal late results led us to look upon blind reduction and natural impaction somewhat askance. Patient discomfort and occasional slipping of such devices as plaster head caps discouraged our use of these appliances. We decided to find some method of reduction which, in our hands, would assure correct anatomic position and some comfortable and stable means of fixation.

Our present attack upon complicated fractures gradually developed as an outgrowth of our treatment of zygomatic fractures. We noted that several patients in whom blind reduction had been done without difficulty and in whom spontaneous impaction had occurred, in what was judged good position, showed imperfect results on later examination (fig. 1). We were unable to explain whether

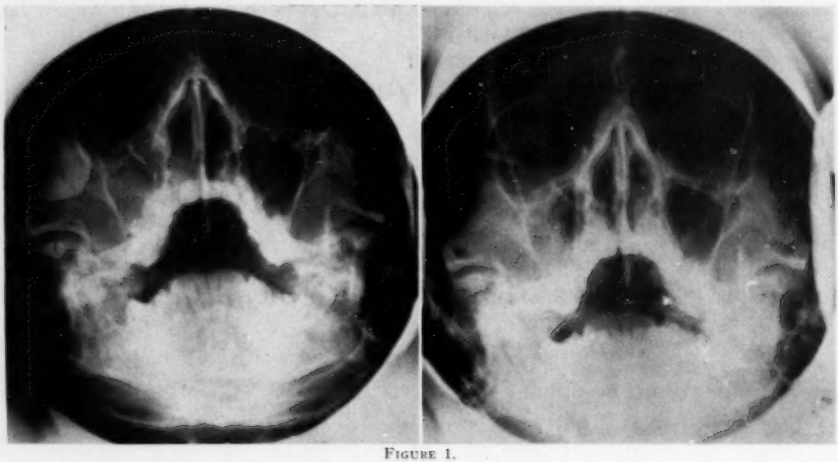

FIGURE 1.

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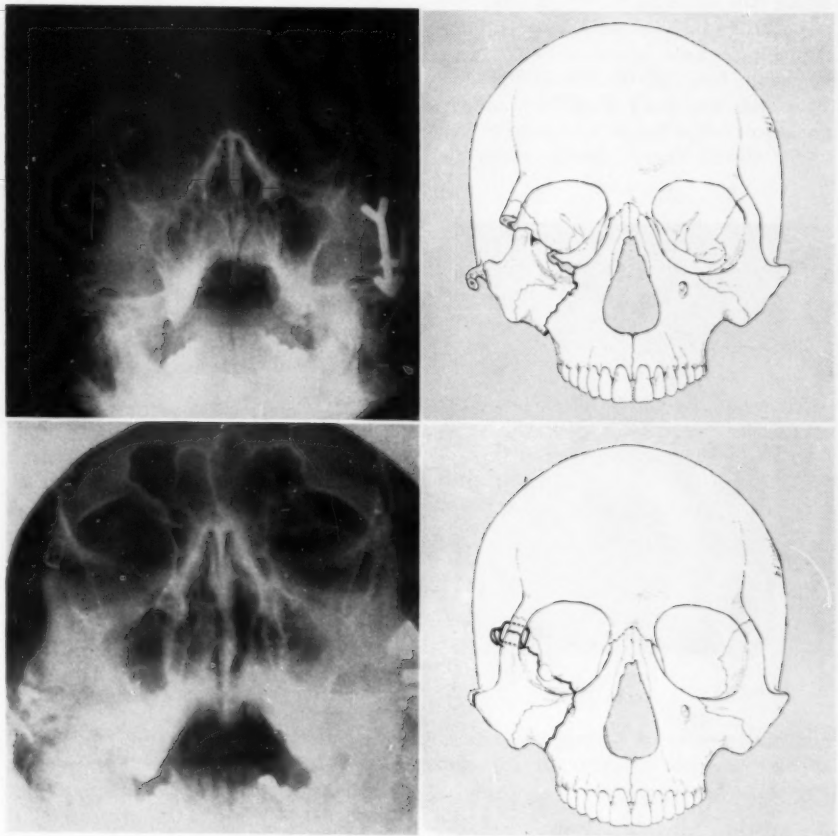

FIGURE 2.

such imperfections were due to inadequate reductions, movement of the bone after reduction, or both. On the other hand, zygomatic fractures which had presented difficulties in reduction or which had failed to impact in acceptable positions had almost uniformly excellent late results. Two factors were conspicuous in the handling of difficult fractures. First, the zygomaticomaxillary and zygomaticofrontal fracture sites were kept under direct vision during reduction.

Extension of the gingivolabial incision across the midline gives direct exposure and allows wiring for multiple facial fractures in which the bones are not excessively fragmented and in which firm points of fixation can be found along the fracture lines (fig. 5).

When treating extensive crushing injuries to the middle of the face it is advantageous to expose the facial skeleton completely through gingivolabial and lateral canthal incisions so that the bony

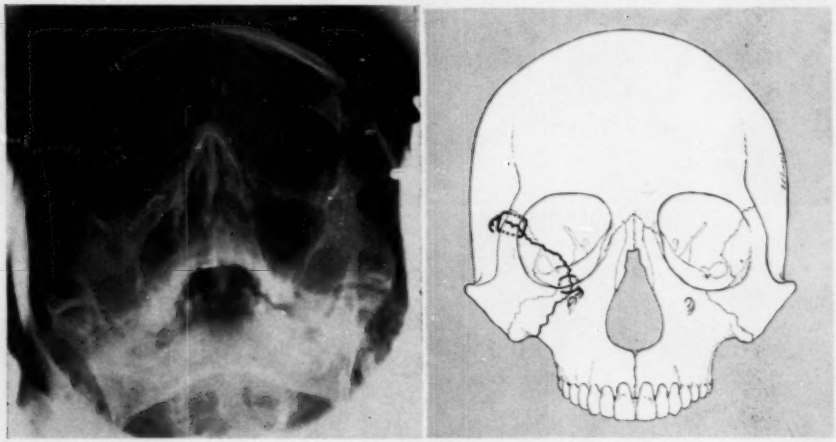

FIGURE 3.

Second, interosseus wiring was done in at least one site to maintain reduction (fig. 2).

Exposure for nearly complete visualization and interosseous wiring of zygomatic fractures can be gained by extending the incision in the gingivolabial sulcus and by making a second incision in a skin crease near the lateral canthus. Soft tissue elevation through either or both of these incisions allows wiring at multiple points (fig. 3). This method is especially helpful when the zygomatic bone is broken into two or more large segments (fig. 4).

fragments can be reassembled piece by piece. The reduced fragments often can be splinted against each other so that individual wiring is unnecessary. If wiring is necessary, the entire mass of crushed bone can be held in position by hammock wires passing from the skull to arch bars on the maxilla or mandible[1] (fig. 6). Intermaxillary ligation can be used to maintain normal relationship between the upper and lower jaws. The even further complicated fractures may require incisions in the eyebrow, across the nasion, in the lower lid, or in the hairline to allow access to bony frag-

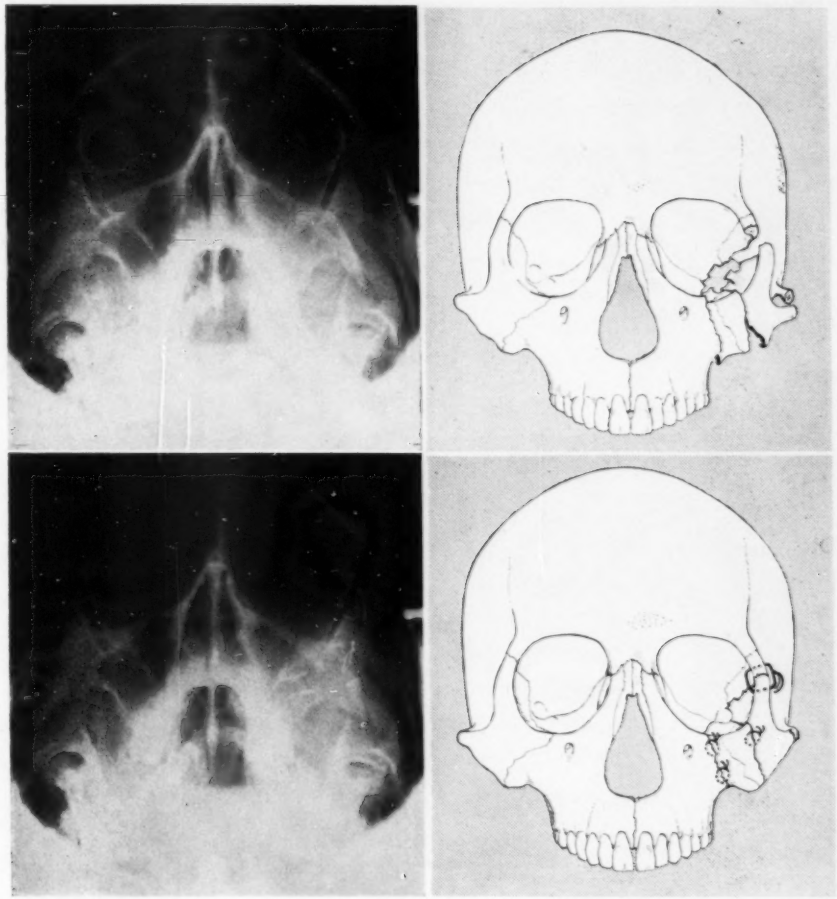

FIGURE 4.

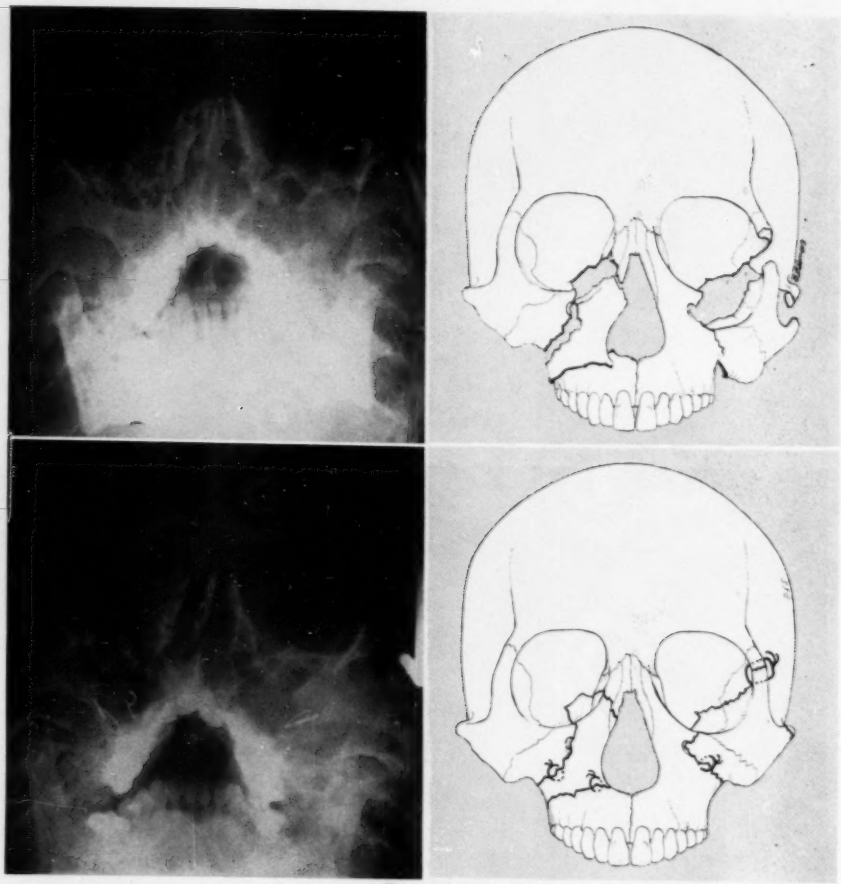

FIGURE 5.

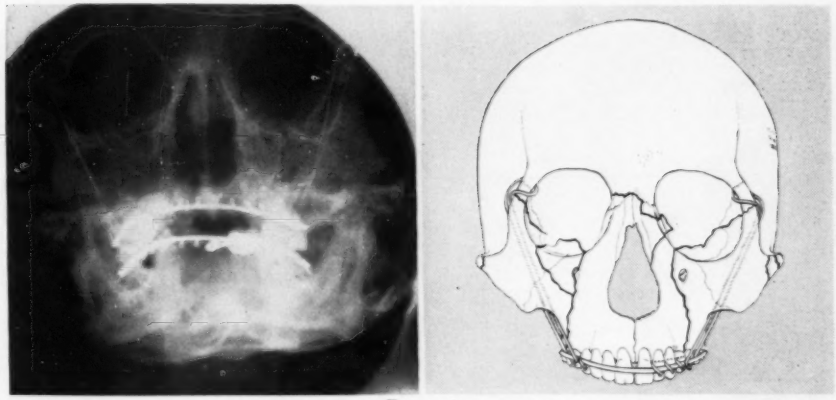

FIGURE 6.

ments and permit direct wiring (figs. 7 and 8).

After we had applied the foregoing principles for several years, we found that Dr. William Milton Adams had antedated our thoughts and experiences by several years.[1] His extensive report on similar methods coupled with our own observations strengthened our belief that open approach and direct wiring offered the simplest and most reliable treatment of complicated fractures.

Nasal damage accompanying midfacial fractures usually can be cared for by intranasal reduction without mechanical fixation. However, if there has been extensive crushing of the bones of the nasal skeleton, some sort of support is required until considerable fibrosis takes place. Although suspension of the depressed comminuted nose by intranasal

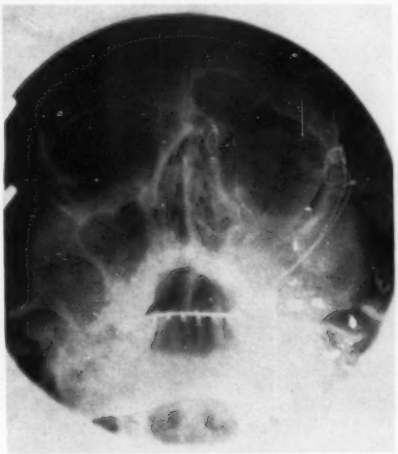

FIGURE 7.

FIGURE 8.

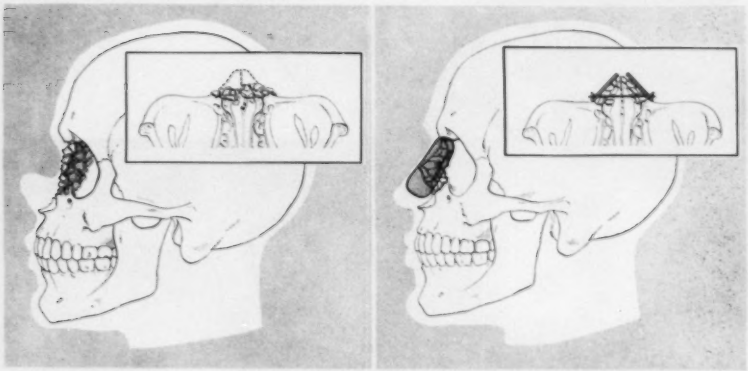

FIGURE 9.

FIGURE 10.

FIGURE 11.

FIGURE 12.

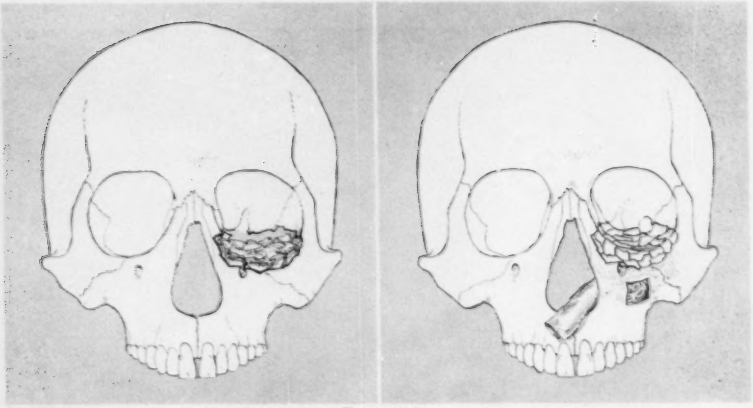

FIGURE 13.

splints and external traction appears to give excellent results in the hands of others, we are hardly ever able to use such appliances to our own satisfaction. In our search for a simple and sure way of caring for the badly crumbled and recessed nose, presenting a greatly increased intercanthal distance, we finally settled upon lateral lead plates with wire suspension sutures passing behind the most posterior available fracture line[3] (figs. 9 and 10). We believe that early narrowing and elevation of the nose is essential. We have as yet found no effective way of dealing with ocular hypertelorism once the fractures have been allowed to heal in faulty position (fig. 11).

Although we try to use the skull or jaws as the fixed points of immobilization or traction, there is one situation in which we have not been able to avoid the use of a head band or plaster cap. The recessed maxilla that cannot be manually reduced because of firm impaction or fibrosis forces us to rely on

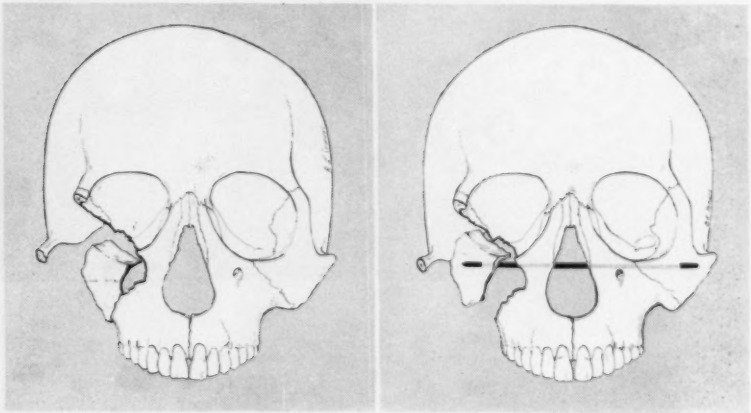

FIGURE 14.

some form of elastic traction; we have been able to gain this only by means of an external appliance (fig. 12).

There are two other conditions in which we are unable to employ interosseous wiring alone. A situation in which bone (usually of the orbital floor) is so finely crushed that wires cannot be passed through the separate fragments forces us to rely upon admittedly makeshift splinting by firm packing (fig. 13). Direct wiring is often impossible when so much bone has been lost by trauma or debridement that bone to bone contact is unattainable. Here we have nothing better to offer than pin suspension of any available fragment from the most accessible solid point[2] (fig. 14). Suspension of fragments in instances of excessive loss of bone is usually done with the expectation of later contour improvement by skeletal grafting.

SUMMARY

All facial fractures need not be treated by open exposure and interosseous wiring. We do not claim that all such fractures can be handled by wiring without the use of external appliances or pins. However, we believe that open operation and bone to bone fixation are relatively simple procedures which can produce satisfactory results in a great number of cases.

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PRINCIPLES OF TREATMENT OF THE FRACTURE OF THE MANDIBLE

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BY INVITATION

FUNDAMENTAL principles in the treatment of fracture of the mandible include:

1. Restoration of the fragments to their correct position.
2. Maintenance of the fragments in their correct position until clinical union has occurred.
3. Prevention of infection.
4. Gradual rehabilitation of function to ensure that subsequent displacement does not occur. Muscle wasting does not take place during the period of immobilization, since some degree of muscular contraction results from speech and deglutition.

Restoration of the fragments to their correct position may be brought about by (a) manipulation; (b) gradual traction (usually elastic bands); (c) open reduction; or (d) skeletal fixation.

The simplest and most easily applied technique that restores normal function should be the treatment of choice. I should like to stress the importance of the surgeon's being most familiar with the proper occlusion of the teeth; in any fracture of the mandible, the teeth, when they are present, serve as a guide for correct alignment of the fracture parts. I quote from Rowe and Killey's "Fracture of Facial Skeleton":

> Elsewhere in the body precise alignment of the fragments, whilst desirable, is not essential, but the slightest error in reduction in the case of fractures of the jaw leads to serious derangement occurring in the occlusion of the teeth, with a corresponding impairment of masticatory efficiency. In the case of fractures of the edentulous jaw some degree of latitude is permissible, as errors in alignment may be compensated when new dentures are ultimately fitted.

Malocclusion may also be a contributing factor in disturbances of the temperomandibular joint.

Teeth also serve as excellent anchorage for wiring and splinting, when these simpler techniques are adequate, and they should also be utilized when more complicated methods are indicated.

Many facial injuries are so severe that the primary consideration is the assessment and treatment of the patient's general condition. The maintenance of the airway, the arrest of severe hemorrhage and the treatment of shock are instituted in this order. In severe shattering of the continuity of the mandible, the tongue may drop back against the posterior pharyngeal wall, thereby embarrassing the breathing. Methods to keep the tongue forward must be instituted at once, particularly if the patient

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is unconscious. It may become necessary to pass a nasopharyngeal tube for the maintenance of airway. Pressure packings will usually suffice for the arrest of hemorrhage. Antibiotics and antitetanus serum should be administered.

Fractures produced by gunshot wounds and those produced by severe traumatizing forces will produce a shattering of the bone. These wounds are debrided in the accepted manner. All debris, shattered teeth and gross pieces of bone that have no attachment to the soft tissues are removed. Those pieces of bone, however small, that remain attached to the soft tissues are to be preserved, for they act as islands of bone regeneration in healing and may be most helpful in preserving the continuity of the bone.

Teeth in line of fracture have always presented a problem and an argument. Our policy is a middle-of-the-road one. If the tooth has been fractured and its removal can be accomplished without complicating the case, then it is best to remove it. If the tooth is in the posterior fragment, near the angle, its removal may be difficult and it may convert a simple fracture into one of gross displacement which may be difficult to reduce. If there is danger of infection, the fractured root may be better removed at a later time when some union has taken place. With the use of antibiotic therapy the calculated risk of leaving teeth in line of fracture is not as great as it was previously.

Reduction of the fracture should be accomplished as soon as the patient's condition will permit. Fixation of the fracture parts either by wiring, splinting, or by dressing, plus early repair of soft tissue injuries, should take priority over prolonged procedures.

Clinically, fractures of the mandible are usually quite evident, for they are usually accompanied by crepitus, malocclusion, inability to open or close the jaws, deformity and pain. These findings should be confirmed by adequate roentgenograms, which may also reveal additional injury, for fractures of the body of the mandible are frequently accompanied by condylar fractures. Roentgenographic studies should be made of both sides of the mandible, with the head in the Water's position, for proper diagnosis. Dental radiograms and occlusal films are of great diagnostic value in fracture of the alveolar process and the symphysis.

No mention need be made of the extent of the part played by muscles of mastication in the displacement of the fragments of the fractured mandible, for they have been amply covered by Dr. Shapiro.

The simplest method, and one that has been so helpful in many cases where there is an adequate complement of teeth, is the Ivy loop method, first described and utilized by a distinguished teacher, Dr. Robert H. Ivy. The occluding teeth in the opposing jaw act as a guide in the proper reduction of the fracture. It is surprising how very versatile this method can be even in difficult cases. Usually this method of reduction can be accomplished under local anesthesia. If general anesthesia is employed, it is our practice not to tighten the intradental wires until the danger of nausea has passed, for if the teeth are wired into occlusion there is danger of the vomitus being drawn into the lungs.

The next method in order of simplicity, and one that is most helpful in those fractures where the displacement is marked and offers resistance to proper reduction, is the arch bar and elastic traction. This, too, can only be employed where adequate dentition is present. The bar is quite easily bent to fit the arch and is wired to the individual teeth. This adds some stability to the appliance. It

is also helpful in bridging edentulous areas. Elastics can be placed to exert traction in any direction desired, and the constant pull of the elastics so fatigues the muscles that the displaced fragment is brought quickly into proper position. This type of fixation was used widely during the war because of its safety factor in sea or air transportation, in which nausea had to be considered. Either the removal or cutting of the elastics gives an easy method of quickly opening the jaws. The Ivy loop or elastic traction will suffice in most fractures of the mandible when an adequate number of teeth are present.

So far we have spoken of the Class I fracture, in which each fragment contains enough teeth for wiring.

In Class II fractures, those in which there are teeth in one fragment only, one is confronted with a different problem. If there is no displacement, this type of fracture may be treated as a Class I fracture, and fixation of the mandible to the maxilla will suffice. If there is gross displacement of the posterior fragment, open reduction or the use of external pin fixation is indicated. With either of these methods it is our practice to immobilize the mandible by fixing it to the maxilla, for in our experience neither the open reduction nor the external pin fixation is ample to allow opening and closing of the jaws.

In Class III fractures, the edentulous mandible can be reduced by a splint and circumferential wire, by open reduction, or by pin fixation. A denture may serve instead of a splint. Here again it is our practice to fix the upper to the lower jaw in some manner.

There are many variations to the methods given for the treatment of the fractured mandible — elaborate cast splints, Kirschner wires, head caps with traction — but time does not permit any discussion of them.

At the present time there is discussion about the fractured condyle. Some surgeons believe that when the condyle is displaced, open reduction and wiring are necessary. I feel that the fixation of the mandible to the maxilla will produce a good functional result, though not necessarily a successful result when judged by roentgenography. It is my belief that many fractured condyles have a tentency to correct themselves.

Teamwork on these facial injuries is to be desired. The plastic surgeon, the otolaryngologist, the ophthalmologist, the neurosurgeon, the oral surgeon, the dentist, are all needed to meet that cardinal principle of all treatment, the well-being of the patient.

Fractures of the Facial Skeleton: A Panel Discussion

ORBITAL COMPLICATIONS OF FACIAL FRACTURES

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A LOGICAL way to visualize the problems of the orbit is to begin at the medial canthus and go up over the roof of the orbit to the apex. The medial canthus is in contact with the lacrimal mechanism. In injuries of the medial canthal ligament and fractures of the nasal bone, the medial canthus is displaced laterally and gives the appearance of a widening of the inner canthal angle. The nasal lacrimal duct is usually fractured, resulting in lacrimal stenosis and secondary infection.

Immediately temporal to the lacrimal groove is the inferior oblique muscle. It originates on relatively heavy bone and injury to the origin of this muscle is unusual. It may be displaced. However, the inferior oblique can take a certain amount of displacement without causing any functional disturbance.

In fractures of the inferior rim there is usually a depression of the floor of the orbit with a herniation of the lower contents of the orbit into the antrum. There is usually an incarceration of the inferior rectus and the inferior oblique muscles, which if not rectified eventually results in fixation by fibrosis; an elevation of the globe becomes impossible. A fracture dislocation of the zygoma in the temporal aspect usually does not seriously involve the orbit. It may cause temporary or permanent anesthesia of the infraorbital nerve. It may create some lowering of the lateral canthal ligament, but restoration of the bone to its proper place will rectify that.

The lacrimal gland seldom is involved in fractures of the orbit. It is unusual to see a fracture of the roof cause any disturbance of the lacrimal gland. The superior oblique pulley may be injured in such a fashion as to result in double vision. Fractures of the roof permit a herniation of the brain into the orbit and a ptosis as a result of impingement upon the levator muscle.

As regards the globe itself, visual disturbances may take place as a result of a fracture through the apex and impingement on the optic nerve. Fractures into the cavernous sinus may cause rupture of the internal carotid artery and exophthalmos with neurologic disturbances. The cornea may become lacerated, and hemorrhages into the vitreous, retinal separation, and loss of vision may follow. Now in taking care of a visual disturbance it is necessary to have an ophthalmologist investigate the situation. Management of double vision is usually not begun until after edema and ecchymosis have been absorbed. If after that has taken place, the patient complains of double vision, monocular occlusion is used until sufficient time has elapsed to treat the double vision otherwise.

I feel that an injured eye muscle or a nerve to an eye muscle causing diplopia should be given six months to recover; however, herniation of the infe-

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rior rectus into the orbit should not remain untreated for six months. It should be rectified immediately before fibrosis takes place. In both of these injuries, regardless of whether diplopia is neurologic or due directly to disturbance of the muscle, the opposing muscles have a tendency to contract and shorten. For that reason the diplopia and muscle charts and muscle function should be followed carefully until it is decided that surgical treatment is required.

NEUROSURGICAL COMPLICATIONS OF FACIAL FRACTURES

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ABOUT 25 per cent of the patients referred to hospitals with fractures involving the paranasal sinuses experience rhinorrhea. In such cases, I think that we all agree that the most important first step for the neurosurgeon handling the case is to establish an open airway, whether by intratracheal tube or by tracheotomy.

Then, of course, shock must be treated. Shock is very rare in uncomplicated head injuries. Compound fractures, in which the frontal bone is visible through the skin of the forehead, of course, should be surgically treated as soon as the patient is out of shock. I believe that one should complete the whole procedure, closing any fistula present, and proceed with the debridement and with the repair of the entire wound.

When fractures are not compounded, the rhinorrhea may be the main problem. The rhinorrhea often does not show up for forty-eight hours; at least it may not be recognized. It is probably because the blood and the swelling of the mucous membranes prevent it from appearing. I keep the head elevated to 60 degrees and warn the patient not to blow his nose at all. I clean the nasal passage mechanically, sometimes putting in Vaseline to facilitate the flow of fluid. Unless there is hemorrhage that is going to endanger the patient's life, the rule is never to pack these fractures. Packing the nose in these cases is the worst thing you can possibly do. Of course, the patient should receive antibiotic therapy immediately.

When rhinorrhea is present, you should remember that the patient may be developing a surgical hemorrhage that will not show the general signs of increasing intracranial pressure — slowing of pulse, respiration, and an increase in blood pressure. The drainage of cerebrospinal fluid can decompress the brain while a concealed hemorrhage develops and death may suddenly and unexpectedly occur. The patient should be watched carefully the first forty-eight hours for impending coma and the other signs of increased intracranial pressure.

Roentgenographic studies are not always helpful. As Dr. Shapiro mentioned, there are such thin walls in the ethmoid region, in particular, and there are so many nooks and crannies that often the roentgenograms are not of great value in demonstrating a fracture in this most important region. But I always take them, at least when the patient is cooperative and out of shock. I try to get routine films and oblique orbital views which may show the optic foramena, the orbital roofs, the crista galli and the ethmoids. When one studies these roentgenograms, one should always look for air intracranially. The presence of air means that there is a tear in the dura and the paranasal si-

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nuses. The air may be subdural or subarachnoid, or there may be air cysts in the frontal lobes.

The onset of rhinorrhea may be delayed for forty-eight hours, for two weeks, and sometimes even for months; and the same holds true with meningitis. Meningitis may develop very quickly; it may occur during the course of a few weeks; or it may not develop until years later.

The site of rhinorrhea, of course, is important to the surgeon. If the rhinorrhea is unilateral and the main fracture site is in the region of the posterior wall of the frontal sinus on that side, it is just about certain that one is dealing with a unilateral lesion and a unilateral flap can be turned. When a unilateral flap is turned, it is wise to use a coronal incision so that, if necessary, another bone flap can be turned on the other side.

Bilateral rhinorrhea will occur if there is a tear in the bony septum between the frontal sinuses, or if there is a bilateral anterior fossa fracture. Occasionally, rhinorrhea can result from a fracture into the middle ear, the fluid traveling by way of the eustachian tube into the nose.

There are tests to determine whether the fluid coming from the nose is cerebrospinal fluid. If a half ounce of clear fluid comes from the nose when the patient bends forward, it obviously isn't mucus. We can be pretty sure this is cerebrospinal fluid. Spinal fluid will test positively for sugar. One must remember, however, that if there is an excess of tear secretion, the mucus may react positively for sugar. I have certainly seen cases where I could not tell whether there was an allergic rhinitis or a cerebrospinal fluid leak. When the leak becomes chronic, diagnosis isn't so much of a problem. Another test is to compress the jugular veins, at the same time holding the head forward.

Facial fractures should be treated as soon as edema subsides. I think that the maxillofacial surgeon is justified in reducing the facial fractures as soon as the edema has subsided, say in three to five days. I have never known meningitis to result from this procedure, nor the amount of fluid to be increased. However, if the surgeon attempted, with considerable effort, to disimpact an impacted fracture, the situation would be different. I believe one should wait at least two to three weeks on impacted fractures to see whether the rhinorrhea will subside.

On the other hand, if there is marked comminution in the region of the anterior fossa, and the leak subsides, there is always the great danger of later trouble. I think the patients with severe fractures should be surgically treated. Usually I turn a bone flap across the midline so that I can treat either side. I usually start with the extradural approach and revert to the intradural approach, as a rule. Defects in the bone are plugged with muscle and I close the dura with sutures or grafts.

The results in a large series of cases in experienced hands have been extremely good. Lewin of Oxford, England, who has had a great deal of experience in the treatment of cerebrospinal fluid rhinorrhea, has summarized his results as follows: of 55 patients operated upon, one developed intracranial infection, none died, and there was no recurrence of the leak. Of 26 patients not operated upon, six developed intracranial infection, four of the six died. The leak recurred in two cases.

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PRACTICAL FUNDAMENTALS OF ANATOMY AND PHYSIOLOGY

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THE function of the lacrimal apparatus is twofold: (1) to secrete an amount of lacrimal fluid, mucus, oil and enzymes adequate to secure proper function of the anterior surface of the eye; (2) to carry off both normal and excessive amounts of this secretion.

THE SECRETORY MECHANISM

The secretory mechanism is composed of two sets of glands: (a) primary glands, which consist of the main lacrimal gland and about 50 small accessory lacrimal glands of Krause and Wolfring; and (b) secondary glands, which consist of the mucin-secreting goblet cells of the conjunctiva and the oil secreting tarsal glands and glands of Zeis at the margin of the lids.

The main lacrimal gland is partially divided into two parts by the aponeurosis of the levator palpebrae superioris muscle. The superior or orbital lobe has two to five ducts which descend over the inferior or palpebral lobe and join with six to eight ducts from the palpebral lobe to empty into the conjunctival sac about 4 to 5 mm. above the outer superior border of the upper tarsus. One or two ducts may empty at or below the lateral commissure of the eye. If the superior lobe were removed, the inferior lobe could still function. However, if the inferior lobe were removed, all the ducts would be severed and the remaining superior lobe inactivated. If the ducts were severed above their openings in the conjunctiva, it would appear that the function of the gland would be destroyed. But this may not be true, for some of the ducts may be able to reestablish communication with the conjunctival sac.

The efferent nerve supply of the main lacrimal gland has been fairly well established. It is parasympathetic and arises chiefly in the superior lacrimal nucleus in the region of the glossopharyngeal and facial nerve nuclei. It is transmitted through the following nerves successively: the intermediate nerve of Wrisberg, the geniculate ganglion, the greater superficial petrosal nerve, the nerve of the pterygoid canal, the sphenopalatine ganglion, maxillary, zygomatic, anastomotic branch to the lacrimal branch of the ophthalmic nerve, which is distributed to the gland itself. Interruption of any one of these will alter the flow of tears.

We can conclude from the observations and experiments of many men, from the time of Aristotle down to the present, that the innervation of the lacrimal gland is not always complete at the time of birth. Sjögren[6] found a complete lack of "reflex" secretion in 13 per cent of full-term babies and 37 per cent of premature babies during the first three days. However, none of these babies lacked enough secretion for the

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normal function of the anterior surface of the eyes. This seems to substantiate the hypothesis that the basic supply of lacrimal fluid is from the accessory lacrimal glands and that the main lacrimal gland is a sort of emergency organ which reacts reflexly to sudden change in its environment, either physical or emotional. The relative lack of symptoms of hypofunction following the complete removal of the main lacrimal gland would also bear out this conclusion.

The terms used for variations from a normal amount of secretion are (a) *hyposecretion,* occasionally referred to as *alacryma,* (b) *lacrimation,* which is true hypersecretion, and (c) *epiphora,* which is due to a faulty drainage apparatus. The first of these is readily established by the Schirmer test.[4] In the latter two it may be difficult to tell the difference. However, in lacrimation the result of the dye test is positive (a drop of dye such as fluorescein placed in the conjunctival sac is recovered from the nose in a few minutes). If the result is negative, the diagnosis of epiphora is established. Also, lacrimation usually varies in quantity, but epiphora is more constant.

Parasympathomimetic drugs, such as pilocarpine, and cholinesterase inhibitors, such as acetylcholine, will increase the flow of tears. Parasympatholytic drugs such as atropine will decrease it. The flow of lacrimal fluid tends to decrease in old age.

THE EXCRETORY MECHANISM

The excretory mechanism consists primarily of the upper and lower lacrimal ducts, better known as canaliculi, the tear sac and the nasolacrimal duct, together with parts of the orbicularis oculi muscle which furnish the motor power in propelling the lacrimal fluid through these passages from the conjunctival sac to the inferior meatus of the nose.

The embryonic origin of the lacrimal apparatus is a pouch at the nasal end of the lower lid which marks the position of the facial fissure. An epithelial cord of cells develops from this, extending into the upper lid and down into the lateral wall of the nose. Canalization begins from the upper end of this cord of cells in the early part of the fetal stage and ends at the lower end of the nasolacrimal duct at about the time of birth. Schaeffer[3] and Schwarz[5] found that in about 30 per cent of newborn infants the outlet of the duct was still closed. Cassidy[7] found that in 73 per cent of full-term, stillborn infants the duct was not patent. Apparently the last barrier that canalization of the lacrimal tract meets is the nasal mucosa.

The canaliculi are about 10 mm. long and consist of a vertical part 2 mm. long and a horizontal part 8 mm. long. The vertical part of each canaliculus begins with the punctum, which lies in the apex of the lacrimal papilla (fig. 1). It is about 0.3 mm. in diameter and is surrounded by a ring of connective and elastic tissue and a constrictor muscle. The lumen then widens to form the ampulla, which is 2 to 3 mm. at its greatest diameter. This in turn gives rise to the horizontal part, which is 8 mm. long and 0.5 mm. in diameter. In 90 per cent of cases, both canaliculi join to enter the tear sac (fig. 2) by a single opening slightly posterior and superior to the center of its lateral wall. This opening, the internal common punctum, may be preceded by a dilation called the sinus of Maier.

The tear sac and nasolacrimal duct are anatomically a single structure with a wide fundus above and a narrowing below as it emerges from the osseous nasolacrimal canal (fig. 3). It usually ends at the vault of the anterior end of the inferior nasal meatus but may extend varying distances down the lateral wall of the meatus. In the first instance the ostium is guarded by a fold of mucosa

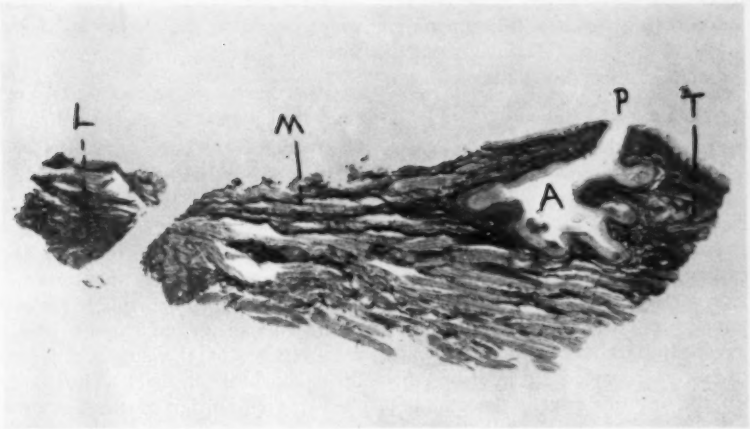

FIG. 1—L—medial palpebral ligament; M—pretarsal muscle, superficial head; P—punctum and verticle part of canaliculus; T—tarsus; A—ampulla. This longitudinal section of the lacrimal part of the lower lid shows that the medial palpebral ligament does not extend to the tarsus.

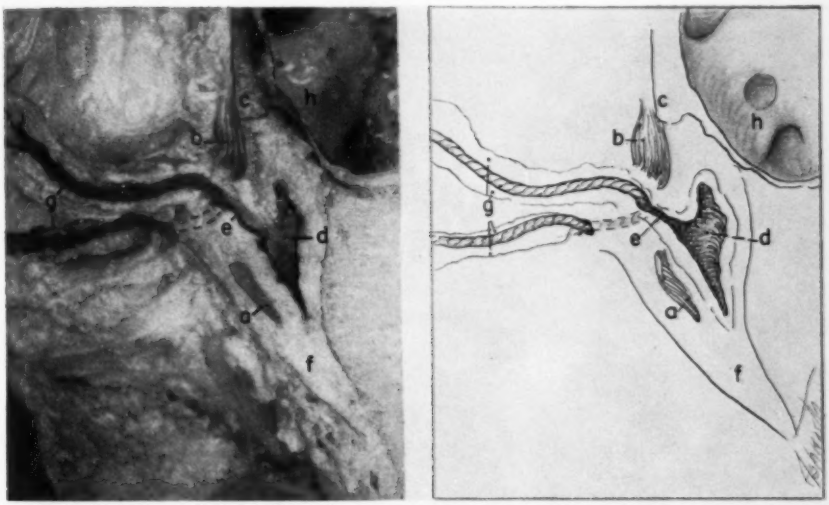

FIG. 2—Horizontal section through medial canthus. a—deep head of upper preseptal muscle; b—Horner's muscle; c—posterior lacrimal crest; d—lacrimal sac; e—sinus of Maier; f—anterior part of medial palpebral ligament; g—black thread in both canaliculi; h—anterior ethmoid cell.

called the valve of Hasner. The second type more often has a slitlike opening.

MEDIAL CANTHAL REGION

Medial Palpebral Ligament

The medial palpebral ligament is essentially a thickening of the periosteum (fig. 4). Its margins, therefore, are not all exact. It begins anteriorly near the nasomaxillary fissure and broadens as it passes laterally to the upper part of the anterior lacrimal crest, where it turns posteriorly to end at the posterior lacrimal crest. It thus has an anterior part and a posterior part. The anterior surface of the anterior part is smooth, being covered medially by the angular head of the quadratus labii superioris muscle and the angular vein and artery. The rest is covered only by skin and superficial fascia. At the lateral end of the anterior part is a 2 mm. projection which gives origin to muscles to be described later.

Most textbooks[1] describe the medial palpebral ligament as extending from the medial canthus to the tarsus of each lid. My dissections fail to show this either grossly or microscopically. If a ligament were present, it would have to pass through one head of the palpebral muscle even to reach the tarsus. The confusion has probably been caused by the resemblance to ligament of the common tendon of the superficial part of the palpebral muscle. The so-called "lower free border of the ligament" is the part which is easily and clearly identified in approaching this region at operation. However, it always becomes muscle medial to the puncta. Interestingly, the old terminology, *tendo oculi*, is more

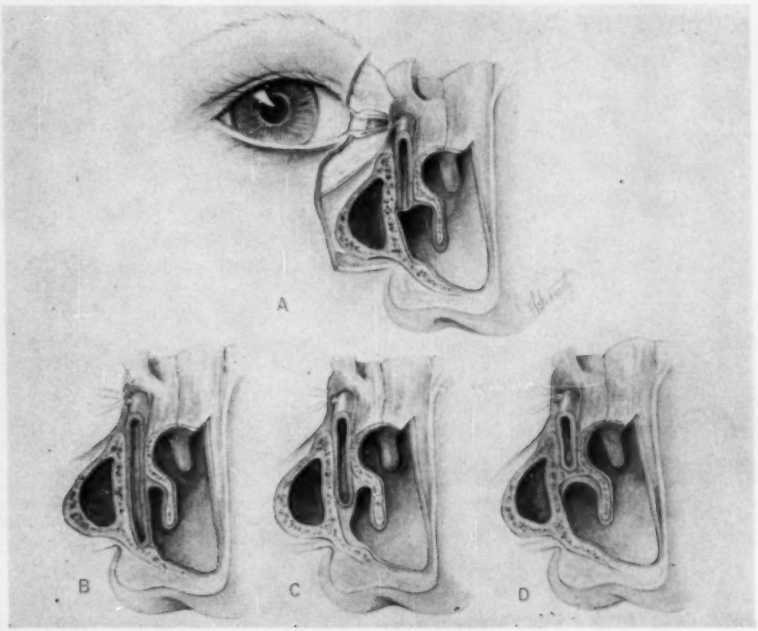

FIG. 3—Various forms taken by nasolacrimal duct. A—normal ending at vault of meatus; B—extension of duct to floor of meatus without perforating nasal mucosa; C—a frequent ending in the wall of the meatus; D—failure of the nasolacrimal canal to form.

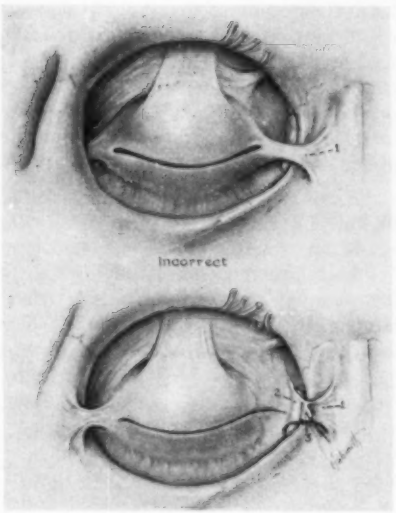

FIG. 4—The medial palpebral ligament. Above—incorrect illustration seen in most textbooks which shows the ligament extending to the tarsi. Below—author's dissections show (1) anterior part of medial palpebral ligament; (2) posterior part; (3) stump from anterior part which gives origin to both superficial heads of pretarsal muscle.

accurately descriptive of this part than is the present terminology.

Lacrimal Diaphragm

The lacrimal diaphragm consists of the extension of the orbital periosteum covering the lacrimal fossa, known as the lateral lacrimal fascia, which is joined in its upper half by the posterior part of the medial palpebral ligament. The lacrimal diaphragm is not only continuous with the periorbita at the margins of the lacrimal fossa, but it is also continuous with the periosteal layer that passes medial to the sac and completely invests it. The diaphragm appears to be thinnest just below the junction of the anterior and posterior portions of the medial palpebral ligament. This would account for perforations that accompany acute dacryocystitis.

Below the level of the posterior part of the medial palpebral ligament, the diaphragm is covered by, or gives rise to, the septum orbitale. This part of the septum orbitale attaches to or is a continuation of the medial ends of the two tarsi. It covers the lateral surface of the deep heads of the palpebral muscle and protects the orbital cavity from extensions of infection from perforations of the sac. Thus, the lacrimal diaphragm converts the lacrimal fossa into a completely closed cavity (fig. 5) except where blood vessels and ducts enter or leave it. It becomes a sac enveloping a sac. A medial movement of the diaphragm creates a positive pressure in the sac and a lateral movement creates a negative pressure.

MUSCLES

Whitnall[8] described the orbicularis oculi as being divided into two main parts—orbital and palpebral (fig. 6).

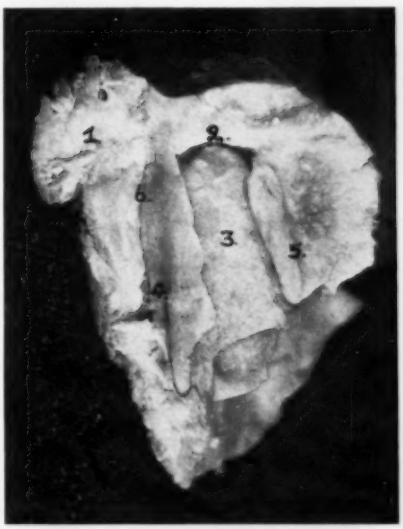

FIG. 5—Medial face of right medial canthal region. 1—anterior part of medial palpebral ligament; 2—upper edge of posterior part of medial palpebral ligament; 3—tear sac showing through a rectangular window cut in periosteum; 4—beginning of a horizontal groove that marks the base of the lacrimal fossa, not visible from this view; 5—groove for posterior lacrimal crest; 6—groove for anterior lacrimal crest.

FIG. 6—Main subdivisions of the palpebral muscle. a and a′—upper and lower pretarsal muscles; b and b′—upper and lower preseptal muscles; and c and c′—orbital part of orbicularis oculi muscle. The dotted line enclosed the anterior surface of the lacrimal pyramid.

We are primarily concerned with the palpebral part, which is subdivided into pretarsal and preseptal. The pretarsal part lies on the anterior surface of each tarsus, the upper being roughly delimited above by the superior palpebral skin fold and the lower by the inferior palpebral skin fold. The preseptal part lies on the septum orbitale between the pretarsal and the bony orbital margin in the upper and lower lids.

The origins of these muscles are at the medial canthus and each has two heads, one superficial and one deep (fig. 7). Each pretarsal muscle is formed by the junction of Horner's muscle (deep head) also known as tensor tarsi [Duverney, 1749], posterior lacrimal, and pars lacrimalis or orbicularis oculi muscle with the superficial head (also known as anterior lacrimal muscle [Gerlach, 1880]) from the medial palpebral ligament. Each preseptal muscle is formed by the junction of a previously undescribed muscle (deep head), which arises

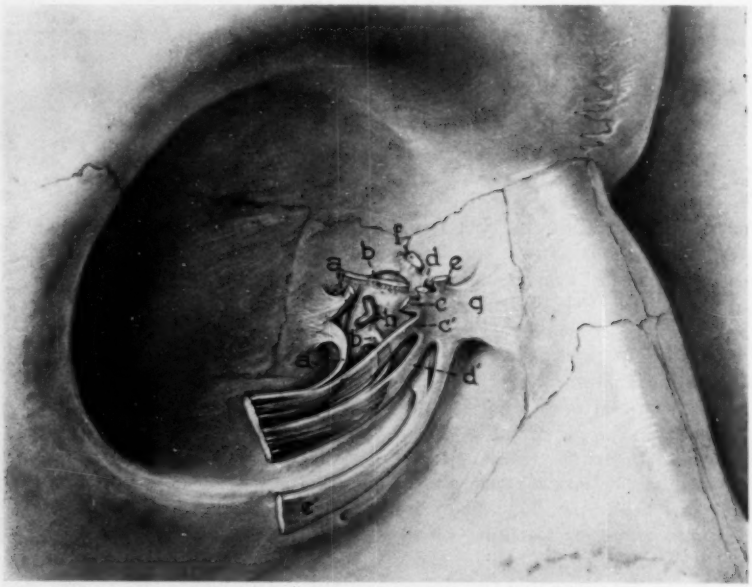

FIG. 7—Muscle origins from medial canthal region. a and a′—upper and lower deep heads of pretarsal muscle (Horner's); b and b′—upper and lower deep heads of preseptal muscle; c and c′—upper and lower superficial heads of pretarsal muscle; d and d′—upper and lower superficial heads of preseptal muscle; e and f—superficial and deep heads of corrugator muscle; e′—lower orbital muscle fibers that arise from the medial palpebral ligament; g—anterior part of medial palpebral ligament; h—junction of canaliculi in lacrimal diaphragm.

from the lacrimal diaphragm, with the superficial head from the medial palpebral ligament.

The anterior part of the medial palpebral ligament gives origin to all the *superficial* heads of the orbicularis oculi (fig. 8). They are arranged in a fanlike series, as follows: (1) from the upper margin are (a) the orbital-corrugator-supercilii and (b) the superior preseptal muscle; (2) laterally, from its junction with the posterior part of the ligament, is the common tendon of the two pretarsal muscles; (3) from the lower border are (a) the lower preseptal, (b) some of the orbital part of the orbicularis oculi, and (c) usually a few fibers of the angular head of the quadratus labii superioris muscles.

Horner's muscles have a common origin just posterior to the upper half of the posterior lacrimal crest (fig. 9). This

FIG. 8—Medial canthal dissection. 1—anterior part of medial palpebral ligament with a lateral extension; 2—for origin of superficial heads of pretarsal muscle; 3 and 3′—superficial heads of preseptal muscle; 4 and 4′—deep heads of preseptal muscle; 5 and 5′—superficial and deep heads of corrugator supercilii muscle.

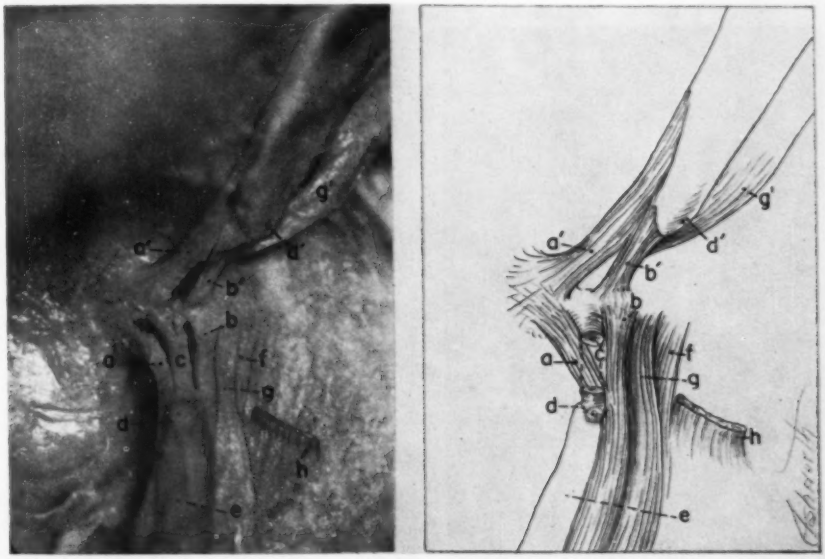

FIG. 9—Medial canthal dissection between upper and lower lids. a and a′—deep heads of pretarsal muscle; b and b′—superficial heads of pretarsal muscle; c—lower canaliculus (cut away in sketch); d and d′—puncta; e—lower tarsus; f—lower orbital muscle; g and g′—lower and upper preseptal muscles; h—angular head of quadratus labii superioris muscle.

muscle helps to form the main body of the pretarsal muscle. Each Horner's muscle is firmly attached by connective tissue to the canaliculus which accompanies it and gives off muscle fibers which insert into it and the medial end of the tarsus. Each Horner's muscle also gives off an isolated fasciculus, the pars marginalis of Riolan, which runs along the palpebral border of each lid.

The deep head of each preseptal muscle arises from the upper half of the lacrimal diaphragm, one above and one below the level of the internal common punctum (fig. 10). Each may be continuous with the origin of the superficial head. The superior is so broad that some of its fibers arise from the lacrimal bone posterior to the posterior lacrimal crest. A pad of fat separates it from the deep head of the corrigator muscle. I believe this is not herniated orbital fat, as has been claimed in the literature, but is a separate pad which prevents friction between the two muscles.

The lateral palpebral raphe is the common insertion for all parts of the palpebral muscle.

The following actions may be attributed to the various palpebral muscle divisions:

1. All are tensor muscles, moving the nasal ends of each lid toward the nose.
2. All jointly retract the globe of the eye.
3. Each pretarsal muscle causes the upper and lower marginal tear strip to flow toward the lacus lacrimalis, squeezes the ampulla shut, and shortens the canaliculus.
4. The upper pretarsal and preseptal muscles are depressors of the upper lid.
5. The lower preseptal muscle is, to some extent, an elevator of the lower lid.
6. The deep heads of the preseptal muscles pull the lacrimal diaphragm lat-

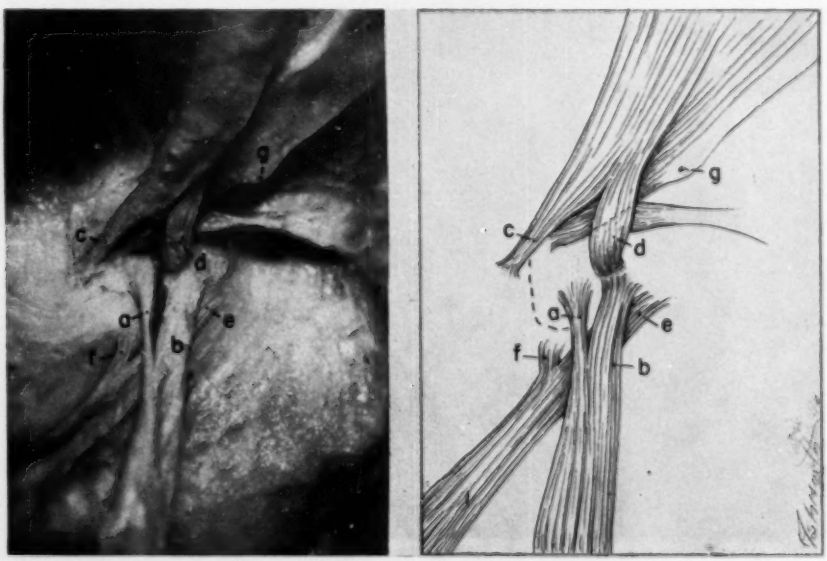

FIG. 10—Medial canthal dissection with pretarsal part of lower lid turned up. a and b—deep and superficial heads of preseptal muscle; c and d—deep and superficial heads of pretarsal muscle; e and f—superficial and deep origins of lower orbital muscle; g—lacrimal papilla and punctum of upper lid.

erally, creating a negative pressure in the tear sac.

The factors involved in moving the lacrimal fluid from its entrance in the upper conjunctival cul-de-sac to the floor of the nose might be summarized as follows:

1. In the conjunctival sac. Tear strips form along the palpebral margin of each lid. These are forced to flow medially not only by the medial movement of the lids but also because, in blinking, the lids close progressively from lateral to medial.

2. In the canaliculus. Capillary attraction, as well as compression and shortening of the ampulla and canaliculus, conducts the tears toward the tear sac.

3. In the tear sac. Alternate negative and positive pressure produced by movement of the lacrimal diaphragm first draws the tears into the sac and then expresses them into the nose. Negative pressure in the nose during inhalation, snuffing, and gravity are also factors in emptying the sac.

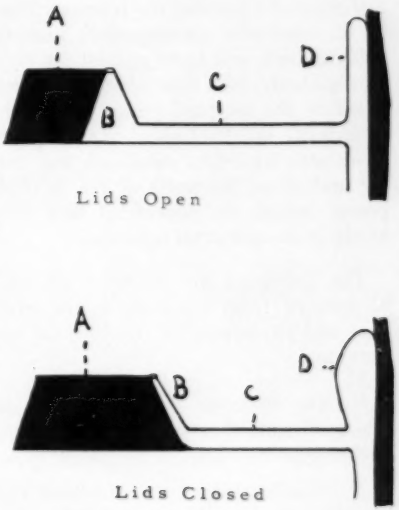

FIG. 11—Diagrammatic drawing of lacrimal pump action. A—tarsus; B—ampulla; C—canaliculus; D—fundus of tear sac.

LACRIMAL PUMP

From the foregoing summary it can be seen that the movement of tears is most dependent on the structures which I have termed *the lacrimal pump*. It is in both lids and consists of (1) the superficial and deep heads of the pretarsal muscles which close the ampullae and shorten the canaliculi, (2) the deep heads of the preseptal muscles which create a negative pressure in the tear sac, and (3) the lacrimal diaphragm. That a negative pressure exists in the sac when we blink is corroborated by clinical observation of mucus in fistulas and intranasal windows which can be seen to recede into the sac during the act of blinking (fig. 11).

As we grow older the forces of ontogeny seem to reverse themselves in the human anatomy. Along with other climacteric changes, especially in the female, atrophy and obstruction may develop in any part of the lacrimal apparatus. Although infection may seem to play a leading role, it is usually secondary to these retrograde, "idiopathic" changes. A wider use of the Schirmer test[4] in elderly people will reveal a surprisingly high incidence of symptoms due to hyposecretion. In the excretory system, obstructions develop most commonly in the nasolacrimal duct. The second point of predilection is in the canaliculus, especially at the internal common punctum. The third is the tear sac itself, in which the cavity may become completely obliterated.

In conclusion may I state that by using the anatomic approach to problems of the lacrimal apparatus we can avoid many failures. For example, we now know why canaliculitis, eroded puncta and more strictures result when we use polyethylene tubing in the lacri-

mal ducts. To prevent the trauma caused by a constantly moving duct mucosa, rubbing back and forth against the rigid foreign body, it is first necessary to immobilize the lacrimal pump during the treatment. We can even offer hope to those who have lost canaliculi and tear sac and all of the parts of the lacrimal pump except the superficial and deep heads of the pretarsal muscles.

The following five points might also be deduced from the study of the anatomy and physiology of the lacrimal apparatus:

1. No ointments should be used in the treatment of the excretory mechanisms.

2. Preservation of the punctum and ampulla is important.

3. The tear sac should not be removed except in case of a malignant lesion.

4. The "pumping" muscles should be carefully preserved during the operation.

5. The prerequisite for the claim of successful surgical intervention on the excretory mechanism is a positive reaction to the dye test.

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THE TREATMENT OF LESIONS OF THE LACRIMAL GLAND

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By no means all of the lesions which arise from the lacrimal gland, or at least in the lacrimal gland fossa, are of a neoplastic nature. Expanding lesions which clinically manifest themselves as well demarcated, localized, firm masses palpable through the upper lid are shown in Table I arranged in order of their frequency according to 112 consecutive cases encountered at the Eye Institute and confirmed by microscopic sections. It can be seen that a chronic granuloma is commonest, and histologically it seems comparable to the so-called "pseudotumors" of the orbit. The adenoid cystic cylindroma, squamous and mucoepidermoid carcinomas, as well as the carcinomas which arise from mixed tumors, are grouped as "carcinoma." The "mixed tumor" has a benign cytology but a malignant behavior due to seeding, invasion of or extension through the capsule, and involvement of adjacent bone. After recurrences, carcinoma may ensue; this tumor is then grouped with the carcinomas. The listed "adenomas" probably represent ectopic lacrimal glands. We must realize that such accessory lacrimal glands may appear anywhere under the conjunctiva (including conjunctiva of the lower as well as the upper lid, the bulbar conjunctiva, and the limbal region). Furthermore, such gland tissue may even appear in the anterior chamber. Therefore, ectopic glands are ubiquitous and must be considered wherever a mass appears.

Tumors found in the salivary gland have their exact counterpart in the lacrimal gland. There is a striking difference, however, in the cure rate of the same tumor in the two different sites. The prognosis for salivary gland tumors is good; that for lacrimal gland tumors is extremely poor. The cure rate of mixed tumors of the salivary glands is 96 per cent, and of the lacrimal gland 50 per cent. Of carcinomas of the salivary gland, the cure rate is 25 to 30 per cent, whereas no carcinoma of the lacrimal gland has ever been reported as cured. The reasons for our failure to treat effectively epithelial tumors of the lacrimal gland seem to be the following: (1) Usually the correct diagnosis is not known when treatment is attempted. (2) Epithelial tumors of the lacrimal gland tend to invade, and even extend beyond, their capsules; or the capsules are missing in places. (3) There is a tendency for the tumor to seed. (4) There is a tendency for the tumor to invade bone, and an ideal opportunity is afforded in the lacrimal gland fossa. The bone invasion seems to be the major factor to account for the difference in the prognosis between these tumors in the two sites — the salivary gland and the lacrimal gland. (5) Lastly, we have an inadequate surgical approach to the lacrimal gland fossa.

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If we review the expanding lesions of the lacrimal gland and its fossa (Table I), we can appreciate the fact that the treatment depends on the diagnosis. If the lesion is a granuloma, one may temporize, because we know that such lesions may show spontaneous regression. If it is a carcinoma, an exenteration is called for, and it is my feeling that the bony orbital wall in and around the lacrimal gland fossa must be resected even if no gross involvement is seen or demonstrated by roentgenologic examination. If the diagnosis is a mixed tumor, the indicated treatment is local excision. If the diagnosis is lymphosarcoma, the treatment is irradiation. There is no means by which a correct diagnosis can be made short of biopsy. Therefore, in our surgical treatment, biopsy is usually necessary. To date I know of no surgical approach to the lacrimal gland fossa which is adequate to handle expanding lesions and tumors in this region. I should like to recommend, therefore, a procedure which seems to meet the requirements better than any that I have employed. It permits the securing of material for biopsy and seems to combat the various causes for failure previously enumerated. This operation has previously been described as follows:

An incision is made in the skin just under the brow concentric with the orbital margin over the region of the lacrimal gland (fig. 1A). The fascial planes over the lesion are incised and reflected. The lesion is firmly grasped with an Allis clamp and a portion is excised (fig. 1A-a), the surgeon taking precautions not to disseminate any of the tissue cells. A deep wedge of tissue must be removed, for sometimes normal lacrimal gland tissue is most superficial. While this biopsy material is being prepared for frozen sections and until the pathology report is given, the fascial planes are closed with plain catgut sutures and the line of closure marked with methylene blue (fig. 1A-c). If the report on the frozen section is "mixed tumor," a modified Krönlein operation is done as described below. If the report is "carcinoma," the skin incision is closed and the patient is informed the following day (or after adequate histologic examination and confirmation), that an exenteration of the orbit is indicated. If the

TABLE I

INCIDENCE OF EXPANDING LESIONS OF THE LACRIMAL GLAND BASED ON 112 CONSECUTIVE CASES

LESION		NO.	%
Granuloma		35	31
Nonspecific dacryoadenitis	26		
Sarcoid	5		
Hodgkin's disease	4		
Carcinoma		27	24
Mixed tumor		25	22
Lymphosarcoma*		24	21
Adenoma		2	2
Total		112	

*It did not seem necessary to divide the lesions listed as lymphosarcoma into reticulum cell, lymphocytic cell, and giant follicle types. Also, lymphomas are included because the demarcation between them and lymphosarcoma is not always clear.

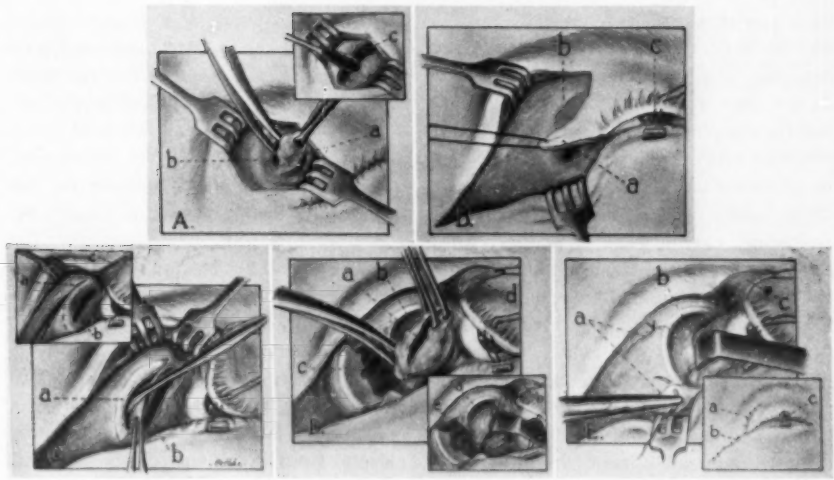

FIGURE 1.

pathologic diagnosis is "lymphosarcoma," then, of course, closure is made and irradiation advised. If the diagnosis is "chronic granuloma," the biopsy incision is closed and the nature of the lesion discussed with the patient. Perhaps no further local treatment is indicated unless the microscopic examination shows a big element of lymphocytic infiltration, in which case benefit might be expected from irradiation.

The modified Krönlein operation is done by extending the incision under the brow to meet the usual Krönlein incision extending horizontally from the external canthus (fig. 1B). The tissues are dissected back to expose the outer half of the bony orbital margin above and the entire lateral wall. In reflecting the lid (in order to expose the upper margin of the orbit) the surgeon must keep his dissection in the plane of the levator muscle. A traction suture is placed in the skin of the upper lid (fig. 1B-a), and the lid is reflected nasally out of the field of operation. An incision is made in the periosteum along the margin of the outer half of the orbit (fig. 1C-insert). With an elevator the periosteum is dissected away from the bone together with the lesion occupying the lacrimal gland fossa (fig. 1C). The lateral wall of the orbit is then resected by using the oscillating Strycker saw and according to the usual Krönlein technique (fig. 1D). The lesion in the lacrimal fossa is then excised together with its adherent periosteum.

The operation can be done without producing a ptosis, or a negligible droop may occur, causing no real blemish.

Exenteration of the orbit is sometimes necessary for neoplasms arising in the lacrimal gland. In this regard I should like to report briefly on the transplantation of the temporalis muscle following the exenteration operation. The usual exenteration, with or without a Thiersch graft, leaves a deep unsightly orbital cavity which, of course, is most objectionable to the patient. Any technique which lessens this mutilation is welcome. If the orbital cavity is partially filled, it not only improves the patient's appearance, but even makes possible the restoration of the orbit and the wearing

of a prosthesis. The temporalis muscle has been employed by plastic surgeons not only to supply dynamic force, such as for the correction of facial paralysis, but for the purpose of using its bulk and vascular qualities. Naquin[6] was the first to advocate use of the temporalis muscle to reduce the size of the orbital cavity following exenteration. Prior to him, Webster[7] had used this muscle to repair a large defect which had resulted from the radical excision of a recurrent mixed tumor of the lacrimal gland. The technique advocated is that of mobilizing the entire temporalis muscle along with its fascia and transplanting them into the orbit through an opening in the lateral wall. This leads to a result more acceptable to the patient. An additional advantage is the fact that no dressings are necessary one week to ten days after the operation. Furthermore, the fact that the skin of the upper and lower lids with their cilia line can be retained makes possible a restoration of the orbit in which a prosthesis might be worn.

I should like now to discuss one of the very interesting new surgical techniques—the transplantation of the parotid duct to the inferior cul-de-sac for the treatment of total xerophthalmia.

This may occur following exfoliative dermatitis, ocular pemphigus, and trachoma, diseases which may cause a complete loss of function of the lacrimal gland and of all accessory gland tissue. As a result, there occurs corneal scarring with marked reduction in vision and most aggravating symptoms. The operation was first advocated by Russians and later by Chinese workers; they reported several hundred cases.[1,3-5,8] There are no reports in the American literature, and the foreign reports give little detail and no appraisal of results. Recently Bennett, and later Bennett and Bailey,[2] have evaluated the operation—first by employing the technique on the dog, then on the cadaver, and finally, on the human. Their approach to the problem, which was reported before the eye section of the American Medical Association in 1957, was most impressive. They performed the operation on one human who had a vision of 12/200 before operation and 20/60 afterward. The flow of parotid secretion into the conjunctival sac was so excessive, particularly when the patient was eating, that it was necessary to do an operation to alleviate the epiphora. This consisted of transplanting the tear sac into the lower conjunctival sac. The parotid secretion closely simulates that of the lacrimal gland and acts as an excellent substitute.

SUMMARY

I would like to emphasize that, of the expanding lesions that occur in the lacrimal gland fossa, approximately one-third are non-neoplastic. Two-thirds are neoplastic but 30 per cent of these (lymphosarcoma) are nonsurgical problems after the diagnosis has been established. Therefore, of these lesions arising in the lacrimal fossa only about one-half require surgical removal. Approximately 50 per cent of those presenting a surgical problem are benign cytologically and 50 per cent are malignant. I have described a surgical approach to this problem of expanding lesions of the lacrimal fossa which I have found satisfactory. It incorporates the removal of tissue for biopsy because the indicated treatment is contingent on the diagnosis. Depending on the biopsy report, there follows either no further surgical procedure, or local excision, or exenteration. If local excision is to be done, a modified form of the Krönlein operation is carried out as described. If an exenteration is to be done, I have advocated the employment of the temporalis muscle to fill the orbital cavity partially.

Lastly, I called your attention to the transplantation of the parotid duct to the lower cul-de-sac for the relief of xerophthalmia.

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ABNORMALITIES AND TREATMENT OF THE PUNCTUM AND CANALICULUS

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DISORDERS of the punctum and of the canaliculus occur frequently, and neglect or improper care of abnormalities of the lacrimal apparatus may lead to annoying epiphora. As epiphora may be difficult to correct, any discussion of this subject should include its causes, its treatment, and, of course, measures for its prevention.

Although in many instances epiphora can be corrected by minor office procedures, the age of the patient should gauge the extent of treatment for disorders of the lacrimal system. Generally, the younger the patient, the greater the indication is for complete repair. In an older person, tear formation usually is decreased in quantity and less epiphora occurs with obstruction of the canaliculus. Therefore, extensive plastic repair seldom is indicated in an elderly patient.

Because time prohibits a description of all of the plastic procedures for correcting disorders of the punctum and of the canaliculus, only a few of the more useful ones will be discussed.

CONGENITAL ANOMALIES

Congenital occlusion of the punctum occasionally may occur. This anomaly may appear as a thin membrane that needs only to be punctured to afford complete relief. Congenital ectropion of the punctum appears rarely; although the degree of ectropion is small, annoying epiphora results. When placed on the inner one fourth of the eyelid, cautery puncture points of the palpebral conjunctiva frequently will cause inward contracture of the margin of the eyelid. If this procedure does not relieve the epiphora, the three-snip operation is effective.

The *three-snip operation* is simple enough for routine office use. Less than five minutes are required to perform this procedure, and gratifying results can be obtained for a wide range of disorders. In performing the operation, the punctum first is dilated sufficiently to allow a small, straight scissors' blade to pass for 2 mm. The scissors then are rotated medially so that the first snip is made in the palpebral conjunctiva adjacent to and parallel to the margin of the eyelid. It is important not to make this snip over 2 mm. in length. Two additional snips are made, each beginning at the extremities of the first snip. These two snips meet toward the cul-de-sac to form the apex of a triangle. When properly performed, the three-snip operation in no way resembles the outmoded slitting-of-the-canaliculus procedure.

Congenital occlusion of the canaliculus seldom is seen, but the treatment is essentially the same as for complete occlusion from any cause. In reconstruc-

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tion of the lacrimal drainage system as it pertains to the canaliculus, duplication of normal drainage is sought. It should be remembered that the purpose of the reconstructive procedure is to relieve epiphora and not merely to develop a patent drainage channel.

In congenital absence of the canaliculus, the development of a patent channel along the normal site of the lower canaliculus offers the most satisfactory route if the lacrimal sac is intact. To accomplish this procedure, the lacrimal sac is opened through an incision in the skin. At this time it should be determined whether the nasolacrimal duct is open. A sharp-pointed instrument, such as a sharp trocar, is passed from the approximate site of the lower punctum. This is forced medially until it reaches into the lacrimal sac. The channel thus made is dilated further until polyethylene tubing (approximately 1.5 to 2 mm. in diameter) can be passed. A very thin, split-thickness skin graft is taken with a dermatome from the inner side of the arm. This skin is sutured around the polyethylene tube in such a manner that the raw surface is outward. Then the tubing with the skin graft is inserted into the newly formed channel and left in position for about two weeks. Mucous membrane from the mouth is too difficult to secure and too fragile to be suitable for transplantation.

STENOSIS

If the lacrimal sac is present, a split-thickness skin graft, as described for congenital absence of the canaliculus, seems to function best for patients with complete stenosis of the canaliculus. If the lacrimal sac is absent, a channel may be created by placing a graft from the medial aspect of the lower eyelid directly into the nose. A channel can be made by using nasal and bulbar conjunctival flaps. The conjunctival flap should be left hinged at the inner canthus and turned backward through an opening created behind the caruncle and into the nose. A strip of nasal mucosa is dissected and passed through the same opening with the raw surface placed posteriorly and the end sutured to the eyeball.

Partial stenosis of the canaliculus is seen rather frequently in elderly patients. Conjunctivitis of long duration may be a contributing factor, and trauma is a frequent cause. When partial or complete stenosis is limited to the lower punctum and/or the lateral one third of the lower canaliculus, the three-snip procedure is indicated and the results usually are more satisfactory than with dilations.

Frequently even partial stenosis is difficult to treat, and excessive trauma of the canaliculus, especially from probing, may convert partial to complete stenosis. To avoid partial stenosis in an infant, closure of the upper punctum with a truncated punctum dilator while fluid is forced into the lower canaliculus may open a congenitally occluded nasolacrimal duct without undue probing. All probes should be greased before they are passed into the canaliculus. A polyethylene tube, inserted to lie free within the lower canaliculus, sometimes is of value for treating partial stenosis. This tube may be left in position for three or four weeks.

ECTROPION

Ectropion of the lower punctum with resulting epiphora occurs frequently in the elderly person. When of a mild degree and/or when it occurs with stenosis of the punctum, the three-snip operation frequently will relieve the epiphora. As a result of prolonged chronic inflammation, however, the caruncle occasionally may be so enlarged that it mechanically keeps the lower punctum away from the lacrimal lake region. In a condition such as this, partial excision or cautery of the caruncle may help to re-

lieve the epiphora. In the more severe forms of ectropion, the Kuhnt-Szymanowski operation may be advisable. It may be necesary to combine this operation with the three-snip operation or some additional procedure to reduce the size of the caruncle.

In the southwestern portion of the United States, particularly in Texas, Oklahoma, and New Mexico, cicatricial ectropion with resulting epiphora may be precipitated by prolonged exposure to sunlight. Generally, this condition requires free skin grafting for correction. Bright sunlight with high ultraviolet intensity in sunny climates also contributes to the frequent development of basal cell epithelioma on the skin surface of the eyelids. Persons with certain skin types are more likely to develop cicatricial ectropion and basal cell epithelioma; and usually persons with light complexions are the most susceptible. In treating a benign lesion or basal cell epithelioma in the region of the punctum and the canaliculus, irradiation therapy seldom is justified. Stenosis of the canaliculus, which may be very difficult to correct, may result from such irradiation. If irradiation is employed, as in treating squamous cell carcinoma, the patient should be advised that epiphora may develop.

PREVENTION

Because epiphora resulting from stenosis of the canaliculus and/or the punctum is so annoying to the patient and at times so difficult to correct, it is fortunate that this is an eye condition in which some of the causative factors can be controlled. As an attempt to prevent epiphora, the following measures are recommended:

1. Gentleness in any manipulation of the canaliculus, including probing, is urged. If handled gently, the canaliculus can be dilated two or three times its normal diameter without noticeable harm.

2. In treating lesions, especially tumors in the region of the punctum and the canaliculus, care should be exercised. Annoying epiphora may be stimulated by irradiation or by traumatic injury during careless treatment of benign, symptomless, new growths adjacent to the punctum.

3. An injured lower canaliculus should be repaired meticulously. This caution may contribute greatly toward the patient's future comfort.

4. Ectropion of the lower punctum as a result of surgical treatment of the lower eyelid should be avoided by allowing for postoperative cicatricial contracture. If there is danger of ectropion following surgical treatment or injury to the lower eyelid, the patient should be instructed to massage the eyelid.

5. To avoid cicatricial changes in the lower eyelid and perhaps in the lower punctum, patients should be warned against excessive exposure to intensive sunlight over prolonged periods of time.

6. Chronic irritation from infections such as conjunctivitis and dacryocystitis which, at times, may result in stenosis of the canaliculus should be controlled. Also, prolonged use of drugs such as Furmethide Iodide and silver nitrate may create irritation and cause stenosis.

ABNORMALITIES AND TREATMENT OF CONDITIONS OF THE LACRIMAL SAC AND DUCT

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ABNORMALITIES of the lacrimal sac and duct may be classified into several groups: developmental, inflammatory, traumatic, mechanical and neoplastic. The lumen of the nasolacrimal duct, although supposed to be uniform, straight and cylindrical, is more often irregular and tortuous. Valves and diverticuli of the lacrimal duct make it uneven, unlevel and hummocky, causing retention of infectious material. These diverticuli develop from auxiliary buds of the rudimentary epithelial cord, which canalizes in segments to form the nasolacrimal duct. External fistulas of the lacrimal sac found below the internal palpebral ligament are congenital diverticuli-like outpouchings which have reached the skin. The lacrimal sac and duct may be crowded by anomalies of the ethmoid cells.

The nasal ostium, located at the highest part of the inferior meatus, just back of the anterior point of attachment of the inferior turbinate to the nasal wall, is usually gaping and wide open. But anomalies of the ostium are common. It may be slitlike, titlike, grooved, closed, or so small as not to be able to function. It may be high, low or at the middle of the wall of the inferior meatus. Its location and character play an important role in the functioning of the nasolacrimal duct and its obstructions. The high incidence of dacryocystitis of infancy (5 per cent) is due to the fact that the lower end of the duct is too frequently not patent at birth and that it does not open spontaneously in all infants but remains closed, retaining the tears.

Congestion or obstruction of the ostium or mucosa of the lacrimal duct may be associated with disease of the nasal or sinus mucosa. Injury to the facial or nasal bones around the lacrimal passages can cause their constriction or obstruction. Rupture or drainage of abscesses of the lacrimal sac may establish a draining fistula. Acute dacryocystitis and rupture of abscess of the sac are virtually diseases of the past, for they respond readily to systemic antibiotic therapy. Concretions of the sac, duct and canaliculi are usually due to actinomycosis infection. Other fungus infections of the sac have been very exceptional. Tuberculosis of the sac during recent years has rarely been observed. Syphilis can induce periostitis or destruction of the nasal bones and obstruction of the adjacent lacrimal passages. Tumors of the sac are uncommon; they may be granulomas or polyps if benign, or epitheliomas or sarcomas when malignant.

Stricture or obstruction of the lacrimal passages may be catarrhal, fibrous, or bony and may occur at one or several points in the lacrimal duct. If epiphora is not from ocular irritation, an attempt should be made to find the site of em-

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barrassment of the lacrimal passages. If a colored collyrium such as argyrol or fluorescein is instilled into the conjunctival sac, the relative rate of disappearance of the color from the eyes may be determined, as well as the ability of the patient to recover the collyrium from the nose. If the lacrimal sac is distended, pressure over the sac will cause mucus, pus, or clear watery fluid to run back through the canaliculus, or the patient will perceive that fluid passes into the nose. If the expressed fluid is clear and thin, obstruction may exist without infection. If the sac is extremely distended and infected, it is unlikely that probing the duct will clear it. Even though no fluid can be expressed and the sac is not distended, nasolacrimal obstruction may be present.

The sac may be irrigated with normal saline solution, boric solution or water, but any solution used should be sterile to guard against infection of normal tissues. If the irrigation of the sac forces fluid back through the canaliculus, the character of the fluid should be noted. If the irrigating solution passes readily into the nose, a functional obstruction may still exist and tears may not drain spontaneously. This type of impediment may be confirmed by roentgenograms taken after instillation of radio-opaque substances such as Pantopaque. Demarest and Milder suggested that more roentgenograms be made a half hour later to determine whether or not the lacrimal passages empty normally.

Dacryocystitis of infancy is well managed by early probing and irrigation of the sac and duct. In small infants this is accomplished without anesthesia as an office procedure. The age of the infant is no deterrent to the probing. In a large number of infants, the lacrimal duct is not patent into the nose. The infant in whom the duct does not open spontaneously may develop intractable dacrocystitis. The probing of the lacrimal duct of an infant, if carefully done, is without danger. If the children are too large to manage without anesthesia, chloroform anesthesia is used in the office.

In probing the duct of infants or adults, the punctum is first dilated, after conjunctival instillation of an anesthetic solution. A syringe fitted with a straight 23 gauge lacrimal cannula and containing 2 cc. sterile normal saline solution is used both for irrigation and probing. After dilating the punctum, a local anesthetic is injected into the canaliculus and into the sac of adults. A small lacrimal probe or the cannula is introduced through the punctum and then turned 90 degrees to a horizontal position as it is pushed nasalward. The canaliculus is stretched laterally to prevent the probe pushing against a fold of mucosa. If difficulty is experienced in probing through the lower canaliculus, passage through the upper canaliculus is attempted in the same manner. When the probe reaches the sac, contact with the bony wall of the lacrimal fossa is experienced. The probe is then elevated vertically with its tip hugging the wall. It is directed downward and slightly backward into the nose. Normally the lacrimal sac has a lumen of about 3 mm. but it may expand because of pathologic conditions. The lumen of the duct is larger, about 5 mm. The duct is directed slightly backward (12 degrees) and inward (9 degrees). Large probes are usually not used as they may traumatize the mucosa. A single probing of the duct may clear the obstruction in adults, but usually does not. The probing is repeated weekly if epiphora persists. After several treatments, if relief is not obtained, dacryocystorhinostomy is suggested.

If a tear sac infection is associated with corneal ulceration, extirpation of the sac is advised. Preceding intraocular surgical intervention, an infected tear sac may need to be removed. Occasion-

ally, in very old or severely debilitated individuals, dacryocystectomy is recommended as treatment for chronic dacryocystitis. It is also recommended for tumor of the sac and tuberculosis of the sac, and it is performed by some surgeons who prefer to avoid the difficulties of dacryocystorhinostomy on patients with so little sensitivity that they would not appreciate its advantages.

Before dacryocystectomy, the lacrimal crest is located by palpation, for it forms the anterior border of the lacrimal fossa which houses the lacrimal sac. The skin incision parallels the lacrimal crest and is medial to the angular vein. The incision is started 2 to 3 mm. above the internal palpebral ligament and is made long enough to expose and deliver the sac. It is deep enough to incise the periosteum of the frontal process of the maxilla, and is made as close as possible to the internal canthus. The periosteum and sac are exposed with a periosteal elevator; the fundus of the sac is loosened of its dense strands and released from its adherence to the lacrimal fossa. The remainder of the sac is freed, its canaliculi cut, the nasal duct exposed and the mucosa of the duct severed as low as possible. A probe is passed through the duct into the nose, and the duct's upper end is curetted to prevent mucosal secretion. The disadvantage of dacryocystectomy is the tearing which remains after lacrimal sac extirpation.

A lacrimal fistula is best dissected and removed down to the sac; then the tract of the fistula is cauterized. If the fistula is associated with chronic dacryocystitis, extirpation of the sac may be required. When it is a small congenital fistula unassociated with sac infection, it will heal if the fistulous tract is removed in its entirety and the tract cauterized.

Dacryocystorhinostomy is advisable in cases where chronic lacrimal sac infection persists after repeated probings. Mosher, Weve, Blaskowicz, Gifford, Iliff, and others have modified Toti's dacryocystorhinostomy. The technique I prefer is that of Dupuy-Dutemps, suturing flaps of the nasal mucosa to those of the sac mucosa to establish a lumen between the sac and nose. The incision of the skin over the lacrimal crest begins at the level of the internal canthal ligament posterior to the angular vein. A cocaine-adrenalin soaked cotton pledget is previously inserted high up into the nose in front of the middle turbinate to anesthetize and decongest the nasal mucosa. The incision is deep enough to incise the periosteum of the crest and is long enough to expose the sac and adjacent lacrimal fossa. After exposing the nasal side of the sac, a lacrimal probe is inserted through the canaliculus into the sac to mark the site of the desired trephine opening. The Strycker saw with its lacrimal trephine is utilized to form the bony opening and to prevent injury to the nasal mucosa. After completing the trephine opening, Kerrison forceps are employed to extend the osteal opening downward toward the nasolacrimal duct. The nasal mucosa is incised vertically and the sutures are inserted at its lower and upper end before the superior, inferior, and horizontal incisions are made. A probe is now placed again through the canaliculus into the sac to mark the site for opening the sac. The incision of the sac on its medial aspect extends downward to the duct and parallels the incision in the nasal mucosa. Care must be taken that the mucosal lumen of the sac is opened, not just its submucosa. The incision of the sac is similar to that used on the nasal mucosa, a large capital *I*. The incisions form a posterior and anterior flap of sac mucosa. Four-zero chromic catgut sutures with small needles are used for suturing the posterior and, later, the anterior flaps. Be-

cause of the small deep opening in which it is necessary to work, the needle must be small (Ethicon G-2). Instead of a needle holder, a small hemostat is used to prevent rotation of the needle.

After suturing the posterior nasal and lacrimal mucosal flaps, the anterior ones are sutured. Following the tying of the anterior sutures, it is best to secure them to the periosteum or bring the needle through the skin to tent the sac lumen forward to prevent collapse. Before closing the wound, the sac is irrigated to be sure that the dacryocystorhinostomy is patent and that the passage into the nose is patulous, not opening into ethmoid cells.

The complications encountered in dacryocystorhinostomy are (1) excessive hemorrhage, unless the angular vessels are watched for, (2) troublesome nasal bleeding at operation or postoperatively, (3) tearing of the nasal flap as the bone is trephined or suturing attempted, (4) necrosing or scarring of the nasal bones or mucosa to prevent their normal function. The failures of the operation are practically always due to faulty surgical technique. If the dacryocystorhinostomy does not function, a second operation to determine the trouble is not difficult because the mucosal bridge and osseous opening have already been constructed.

Where repeated surgical procedures do not produce a patent lumen, Summerskill's flanged polyethylene tube can be inserted into the nose through the osseous lumen. This tube is 1 cm. long and has a lumen of 2.92×3.73 mm. in its inside and outside diameters. These flanged tubes have been used with anticipation that they would remain in position. Frequently, however, they are lost into the nose. It is suggested that they be sutured into place.

I have used the Toti-Mosher operation, Gifford's technique of putting the lower end of the sac and duct into the nose with silk sutures brought through the new bony opening, but I have had better success with the Dupuy-Dutemps technique. I have kept polyethylene tubes in place, strapped to the face, for months. I have at times cut polyethylene tubes flush with the canaliculi, and have lost two of these into the sac. In my hands such tubes incite canaliculitis, thus aggravating rather than relieving the symptoms. Since the lacrimal passages begin in the eye and end in the nose, they are best managed by a degree of teamwork between the ophthalmologist and rhinologist. If nasal obstruction or sinus infection exists, neither probing nor dacryocystorhinostomy will relieve the obstruction until the nasal disease has received adequate attention.

DISCUSSION

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DR. BEARD: This has been a most valuable symposium. Tearing is one of our most difficult problems and certainly one which we often handle not too well.

I have listened with interest to Dr. Jones' accounts of careful dissections and his interpretation of them. I have also read his previous publications and have been very much impressed. Certainly his description of the lacrimal pump has done much to clarify our ideas as to how the tears get from the eye down into the nose. There is one thing that puzzles me now. In the type of dacryocystorhinostomy that I have been accustomed to doing the lacrimal pump is obliterated, or at least immobilized, and these patients do not have tearing. Maybe Dr. Jones can tell me how that can come about.

Dr. Reese's paper on the lacrimal gland recalls his Bedell lecture of, I believe, two years ago, and the emphasis that he placed upon biopsy at the time of operation. I have been impressed with the reluctance of pathologists to give us a final diagnosis on an important thing like this on frozen section. So often we ask for a diagnosis so that we can go ahead with the proper procedure, and the pathologist says that he would rather we would wait for paraffin sections.

Regarding exenteration of the orbit with temporalis flap repair, I have used this procedure on occasion and find that it gives a very pleasing — at least not unpleasing — cosmetic appearance. However, in two cases of exenteration for basal cell carcinomas in which I had filled in the orbit and left a rather smooth cavity, the carcinoma recurred deep in the orbit and went on, unbeknownst to me, to a point where treatment became most unsatisfactory. Since then I have been putting a skin graft directly upon the bone on most of these cases. I believe that the skin graft holds back granulations, holds back the tissue from forming deep in the orbit, and because of it I believe that we can recognize recurrences early, perhaps early enough to treat them adequately.

One or two points on Dr. Vier's paper: I have used the three-slit operation on many occasions and have found in some of them quite a tendency for the opening, which I had felt was adequate in size, to contract until it would not carry away the tears. In a few cases, in desperation, I have resorted to canaliculus slitting and have been pleased and surprised with the result of a nontearing eye and a rather happy patient. I don't

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believe that the operation of canaliculus slitting should be completely discarded, provided that the patient is properly chosen.

I have used split-thickness skin to construct a canaliculus. In one case I could wash through very easily; still there was epiphora. I wondered if it was because of desquamation of epithelium blocking the duct which had been so laboriously made. I wonder if a small dermatome could be made that would allow us to take mucosa from the inner side of the lip. Perhaps a razor blade dermatome could be constructed to allow us to get thin mucosal grafts. These grafts might avert the desquamation which has blocked some of these reconstructed canaliculi.

Dr. Cassady states that acute dacryocystitis is a disease of the past. At least in central California we see a fair number of these cases still. I am sure that I have at least two or three cases a year of acute dacryocystitis which have reached a point, when I first see them, at which incision and drainage are necessary.

With regard to probing in infants, I have always felt more secure when I have given general anesthesia. Regardless of the age of the infant or child, I have an anesthetist come in as a routine office procedure. The anesthetists in our area use Vinethene, enough to quiet the child, and I have felt that that was more satisfactory than wrapping and holding him.

In doing a probing, I feel that it is facilitated by making a slight forward bend in the Bowman probe. Very often the brow gets in the way of the straight probe and will prevent proper angulation in entering the nasolacrimal duct.

I agree with Dr. Cassady that dacryocystectomy is often a good operation. I have heard it said that there are no longer indications for dacryocystectomy, that dacryocystorhinostomy should be used in all cases. I think that in some selected cases dacryocystectomy is the preferred procedure, although often when the sac has become infected and dilated, it is very difficult.

In dacryocystorhinostomy I have used a Stryker saw and have found that, at least in my hands, it is a bit awkward. I have not used the new saw, the small one that has been made for this purpose, but I have had a hard time in guiding the trephine properly into the lacrimal fossa. I have to resort to hammer and chisel very rarely. Usually if one takes a curved hemostat and passes it back into the lacrimal fossa, the bone is very thin and can be broken through very easily to a point where a small Kerrison rongeur can be used to complete the opening. In this way the nasal mucosa is not often cut, at least in my hands, as it has been with the Stryker saw.

DR. SPAETH: There are only two or three things that I want to say.

The demonstration of the lacrimal pump was most ingenious. I am not convinced that the physiologic situation described always exists, because there are repeatedly cases in which one sees the negation of such a possibility, and the best example of them all is the congenital fistula.

The congenital fistula certainly is going to nullify the positive-negative pressure within the sac. Still, as one looks at the child or the infant with a congenital fistula and sees tears forming at the opening of the fistula, one wonders whether pure capillary action of the canaliculus is not equally important.

There is no doubt that when we tear, we blink; so there is a pumplike action of some type. But I don't believe personally that it is only that.

I agree with Dr. Beard; I want to do my probings on infants under general anesthesia. Therefore, I must of necessity disagree with Dr. Viers. I

must also disagree that irrigation should follow probing. I have two reasons, both of them good: (1) The anesthetist won't permit me to irrigate while the infant is under general anesthesia—that is a very good reason, for we don't want the youngster to inspire the fluid. (2) There is the possibility of a cellulitis of the already traumatized nasal lacrimal passage.

Regarding the next point, I quote Dr. Reese's statement in his text on ocular tumors when I state that the surgery of mixed cell tumor of the lacrimal gland is woefully inadequate. The high incidence of mortality demonstrates that conclusively.

Further, if at the time of the operation frozen sections prove the lesion to be adenocarcinoma, I believe one should proceed immediately with exenteration. If it should be a mixed cell tumor of the lacrimal gland and roentgenograms have demonstrated changes in the density of the bone, or if one can see gross changes of bone involvement, then I feel that the exenteration should be done at once. Therefore, the preparation of these patients, as one talks with them prior to the operation, is rather important. I tell the patient that it is quite possible that the exenteration will need to be done at the time of the initial operation.

Nothing was said by Dr. Cassady about malignant lesions of the lacrimal sac. I wish he had considered this topic because the mortality due to malignant lesions of the lacrimal sac is almost as great as that due to malignant lesions of the lacrimal gland. The tragedy is that in a large percentage of instances the diagnosis of malignancy of the lacrimal sac is not made until a sac extirpation or, at the worst, a dacryocystorhinostomy is being done. In those cases in which a malignant lesion is manifest, the situation is different. I am pleading for an operation for lacrimal sac malignancy as radical as that which was outlined by Dr. Reese, and which I have emphasized, for lacrimal gland malignancy.

DR. HUGHES: There are a few aspects of this subject that have not quite been covered. I am going to take a few minutes to discuss some of them.

I know Dr. Reese couldn't discuss the extirpation of the lacrimal gland and the question of dacryoadenitis and its treatment. I think the extirpation of a normal lacrimal gland is indicated in certain cases of excess tearing when nothing else has been done: this is my suggestion for Dr. Viers, who said that nothing could be done for some of these patients with long-standing tearing although the drainage apparatus is perfectly open. I think such an approach is quite feasible, and I have never seen a patient with a dry eye after an extirpation of the main lobe of the gland.

There are a few things about a congenital dacryocystitis that I should like to stress. When you first see a baby with a full sac, massage over the sac will often bring about an opening of the nasolacrimal duct down into the nose. This can be done very readily, by putting the thumb over the medial canthus to block off the punctum and pressing from there over toward the nose and down toward the opening of the nasolacrimal duct.

There are a number of causes for failure in probing these congenital atresias of the nasolacrimal duct. I agree with Dr. Beard when he says that he likes to have the probe well bent. I like to have it bent almost in a half circle; the path it takes forms almost a half circle when it is being used. Injury to the punctum is not infrequent in probing; I think that it can usually be avoided by probing through the upper punctum after having made sure that the lower punctum and canaliculus are open into the sac, either by expression from

the sac or by irrigation at the time of the operation. I make a plea for probing through the upper punctum rather than the lower, to avoid any possibility of injury to the lower punctum. I agree with the statement that one should not irrigate freely after probing. I have seen that done, and I have seen the infiltration of fluid into the tissues with considerable inflammatory reaction, cellulitis with swelling, and so on. I do put in two or three drops of 30 per cent sulfacetimide, following the probing.

In relation to the probing itself, one must be very sure that he is not probing down inside the mucous membrane on the side of the nose. Sometimes the probe will undermine the mucous membrane.

Two or three times I have encountered a little hump of bone down here (slide). It may have been the hump that is shown there, but there was a very definite shelf down here. The probe went down to that shelf. I thought the probe was in the nose, but on putting another probe into the nose, which is routine practice, I could not feel the probe coming from above. This is a good practice. If you feel for the probe coming down through the duct with another probe going backward from the meatus of the nose and touch metal to metal, you will never make the mistake of thinking you have probed into the nose when the probe is, in reality, down inside the mucous membrane or is not down to the floor of the orbit before the opening is made.

(Slide) A very common condition is the eversion of the nasal end of the lower lid that we see frequently in older people. I have found that the Zeigler cautery gives good results in mild cases of this sort, but I do think it has to be very carefully applied, and I think the proper place for it is inside, through the conjunctival surface, just behind the punctum. One puncture is made just behind it; another one, in a vertical row just below that; and then another series of two just nasal to that. If it is a severe case, a couple of punctures are added along a horizontal line. The Ziegler cautery is used to produce a cicatrix which contracts. If you make the cautery points in a vertical line here, you tend to invert the punctum much more than if you put your punctures in at random. I think it should be done specifically to produce an in-turning of the punctum. Of course many of these patients have an atresia of the punctum; correction of the atresia requires the three-snip operation that Dr. Viers spoke about. I think the three-snip operation is an extremely useful office procedure which will relieve many patients of their annoying epiphora.

(Slide) This is a case in which the lower lid had been injured and the punctum had been cut across about a year previously. I was able to dissect the two sections of canaliculus and sew them together in an oblique direction. I think the direction is important. Cut the distal end of the canaliculus in an oblique direction and make a matching symmetrical incision across the other end of the canaliculus; the union is thus not straight across but has an oblique direction. You will find that the canaliculus will probably stay open. For this procedure use extremely fine sutures with very sharp needles.

(Slide) This shows the boy after the repair was done. I placed a polyethylene tube in the canaliculus, sewed in position so it was just barely sticking out of the punctum, and left it in position for one year. He is a very happy boy at this stage, because he is over his tearing.

(Slide) This is a Streptothrix infection of the upper canaliculus; you can see the characteristic bump over the area of the canaliculus and a small amount

of pus coming out of the punctum. These findings determine the diagnosis. In many of these cases, even if the infection is in the lower canaliculus, irrigation can be perfectly done, but the patient will still tear. He will have the characteristic concretions in the canaliculus. I think that under these conditions, the slitting of the canaliculus is in order. Do not slit the punctum. Make the slit medial to the punctum between the punctum and the sac and put it inside on the conjunctival surface. Evert the tissues over the canaliculus and make the incision so that the canaliculus is opened away on the inside, not on the top. If the opening is on top, epiphora will result, especially if the lower canaliculus is involved. Be sure that all concretions are removed from the canaliculus.

Frequently, dilated ampulla will be found in the canaliculus. Be very sure that it is curetted thoroughly, because it may be much larger than expected. Swab the wound with iodine thoroughly. Leave an iodine pledget in place for ten minutes or so to clean the canaliculus thoroughly. Then give some potassium iodide to use internally and also some potassium iodide drops to use locally.

(Slide) This is a characteristic acute dacryocystitis. It is often possible to aspirate the cyst through the punctum itself. Although the sac seems to be entirely blocked off, the punctum and canaliculus may be entered very nicely if they are thoroughly anesthetized. For anesthesia it is best to use a pledget over the punctum and inject some local anesthetic into the canaliculus with a cannula. Replace the pledget every four or five minutes, three or four times. It takes fifteen or twenty minutes to anesthetize the lacrimal apparatus, especially in these cases of dacryocystitis because the tissues are extremely tender. Subsequently, a curved cannula may be gently passed down into the sac, and the pus from the sac aspirated. Then the sac can be flushed with a little sulfacetimide or penicillin.

(Slide) This is a chronic mucocele; the sac is blocked off at both ends. Presence of such a mucocele is not always a contraindication to dacryocystorhinostomy. You can very often feel your way down into that sac, through either the upper or the lower punctum. Use of the upper punctum is preferable in order to preserve the lower one uninjured, since the lower punctum from a physiologic standpoint is much more important than the upper one.

Probing in the office can be done almost painlessly if time is taken to anesthetize the tract properly. Put a pledget over the punctum and repeat that while the anesthesia that is injected down into the canaliculus and down into the sac is acting. Fill the sac with Pontocaine Hydrochloride or tetracaine hydrochloride, or whatever local anesthetic you use and leave it there for five minutes. Repeat this procedure at least three times, and each time replace that pledget over the punctum or the conjunctival surface. If this procedure is followed, probing may be done with very little pain to the patient. If the patient still experiences pain in the nasolacrimal duct area, a little procaine hydrochloride can be injected through the skin into the nasolacrimal canal to anesthetize the duct.

(Slide) I think dacryocystorhinostomy is indicated in 99 per cent of the cases of dysfunction of the lacrimal sac. Of course, if the lesion is malignant or if there is some tuberculous involvement or other granulomatous inflammatory involvement, the situation calls for dacryocystectomy, but I feel that in a great majority of these cases, rhinostomy is the operation of choice. It can be done in elderly people perfectly well. I have done

it in many persons over 70, and in some who were over 80. It can be done under local anesthesia in those cases, but care should be taken to obtain thorough anesthesia. I think that the anesthesia can be made absolutely perfect for the patient, without any pain at all. First, the nose must be sprayed and a cocaine-soaked pledget put up in the region of the sac on the inside of the nose. Then the skin is injected over the site of the incision. The most important parts of the anesthesia are the two deep injections, one at the upper margin and the other at the lower extremity of the sac deeply (about 1.5 cm.) along the nasal wall of the orbit, right next to the periosteum so that it goes behind the sac. The patient will complain of a little pain when the periosteum is reached because the periosteum is sensitive. These two injections are very important.

(Slide) For the third injection, pass the needle along the course of the nasolacrimal duct so that actually you can feel it go down into the nasolacrimal canal; inject about 0.5 cc. there. This operation is absolutely painless, and the injections help in controlling some ot the bleeding that is often an annoying factor.

Often it has been said that when intraocular surgery is foreseen or when corneal ulceration is present, dacryocystectomy should be the operation of choice instead of dacryocystorhinostomy. I heartily disagree with this. It has been shown by laboratory work that the conjunctival sac is more subject to infection and many more infective organisms are present after a dacryocystectomy than after a dacryocystorhinostomy. Dacryocystorhinostomy re-establishes the normal physiologic drainage of the lacrimal sac, while dacryocystectomy only changes the location of the stagnant secretion from the lacrimal sac up to the conjunctival sac.

The age of a patient need not be considered when planning an operation. Dr. Jones demonstrated the means of finding the sac; this is an important point. Many men experience difficulty in locating the lacrimal sac. Make the incision over the nasal attachment of the ligament. Superficial bleeding may be controlled by the lacrimal speculum if the prongs are inserted fairly deep. Locate the attachment of the nasal end of the medial palpebral ligament where it is attached to the periosteum, right anterior to the sac. Detach the ligament from the periosteum and you will find the sac directly underneath. In this way you just can't miss it. If the incision is made close to the periosteum, the sac will not be cut. Then cut the fascia as it is attached to the anterior lacrimal crest and push it and the sac temporally.

In making the mucous membrane anastomosis, if the nasal mucous membrane of the sac has been injured, or if for some special reason a good posterior flap and an anterior flap cannot be made, one good flap across from the sac to the nasal mucous membrane will provide drainage. It is necessary to have at least one good flap, and two are better from the standpoint of drainage.

APPLICATIONS OF HISTOCHEMICAL METHODS FOR THE DEMONSTRATION OF ACID MUCOPOLYSACCHARIDES TO OPHTHALMIC PATHOLOGY

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DURING recent years several special staining procedures, often listed among histochemical methods, have been applied to the demonstration of acid mucopolysaccharides in formalin-fixed, paraffin-embedded tissue sections. Researchers in the laboratory of ophthalmic pathology of the Armed Forces Institute of Pathology have been investigating the usefulness of two of these methods: (1) the Rinehart-Abul-Haj modification[13] of Hale's colloidal iron procedure,[5] and (2) several variations of Steedman's application[15] of alcian blue 8GS.[3,7,10,16,17]

Although acid mucopolysaccharides (AMP) are stained blue with both of these methods, the principles upon which the procedures are based are believed to be different. Hence, positive results obtained by both methods command attention even though neither procedure is, strictly speaking, a specific histochemical test. The value of these stains can be increased by applying them simultaneously to duplicate sections which have and have not been pretreated with hyaluronidase. Only the bovine testicular hyaluronidase commercially available (Wydase, Wyeth) has been employed in our laboratory and since its activity is not entirely restricted to hyaluronic acid,[8,9,11] we cannot conclude that hyaluronidase-sensitive AMP is necessarily hyaluronic acid. Initially the deparaffinized tissue sections were treated for 15 minutes with hyaluronidase prepared by reconstituting an ampule of frozen-dried enzyme in 1 ml. of isotonic sodium chloride solution. Subsequently it was found that treatment of 30 to 60 minutes is often required to abolish the staining reaction of large accumulations of AMP assumed to be hyaluronic acid. Pearse[11] thinks that material in fixed paraffin-embedded tissues, whose metachromasia is reversible by one to three hours treatment with a purified testis extract, is chondroitin sulfate A or C or hyaluronic acid, or a mixture of these.

The observations I am reporting were made on human eyes (usually surgical specimens) fixed in aqueous formalin and embedded in paraffin. Normal eyes were extremely few and were greatly outnumbered by diseased specimens. In many cases, however, enucleation was performed because of a small intraocular tumor or some extraocular lesion; and from such material ample "normal" tissue was available for study. In a recent report based on similar specimens hyaluronidase-sensitive AMP in abundance was described in the intertrabecular spaces and in the iris stroma of eyes with normal anterior segments.[19]

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NORMAL RETINA AND OPTIC NERVE

With the exception of one layer, the normal retina and optic nerve of man fail to reveal the consistent presence of AMP by the techniques employed in this study. The one striking exception is the layer of rods and cones. Although an abundance of hyaluronic acid fills the vitreous chamber and the union between the vitreous and the inner layers of the retina is extremely intimate, I have not been able to demonstrate significantly positive reactions for AMP in the internal limiting membrane and adjacent retinal tissues. The internal limiting membrane and the insertions of Muller's fibers into it frequently give a strongly positive reaction to the periodic acid-Schiff (PAS) procedure, but they are not significantly positive with alcian blue or colloidal iron. This lack of parallelism between PAS-positivity and AMP-positivity has been observed repeatedly by others[1,2,3,6,7,11,17,18,19]; Persson[12] demonstrated that depolymerization of hyaluronic acid gives rise to PAS-positivity.[17]

In sharp contrast to the negative reaction of the bulk of the retina is the intensely positive reaction exhibited by the layer of rods and cones. Although the entire thickness of this layer may be stained intensely, the outer segments often appear to contain most of the AMP. When this layer is examined by means of oil-immersion microscopy, the material responsible for this strongly positive reaction seems to have formed an external coating or intercellular matrix in which the visual cells are embedded. If alcian blue is combined with the periodic acid-Schiff reaction, this relation seems even clearer. While the protoplasm of the visual cells is PAS-positive (magenta), the matrix which lies between these cells and which clings to their surfaces is alcian blue-positive (bright blue). This histochemical characteristic of the rod and cone layer is demonstrated equally well by the two techniques employed in the present study; furthermore, the staining reaction is not altered by pretreatment of the sections with hyaluronidase, even though the enzyme effectively eliminates the stainable AMP from the vitreous, iris, and trabeculae.[19]

To the best of my knowledge, application of these new techniques for the demonstration of AMP has not been described previously in the histochemical study of the retina. Lillie[6] and Wislocki and Sidman[14,18] have made extensive histochemical analyses of the retinas of several animal species, but they utilized metachromatic dyes and various modifications of the periodic acid-Schiff and performic acid-Schiff reactions for their study of retinal carbohydrates. Lillie, working mainly with quick-frozen material, described a distinct violet to red-purple metachromasia of the distal segments of retinal rods when stained with 0.1 per cent thionine buffered to pH 3 or pH 5. Variable results were obtained when fixed tissues were used. The metachromatic material described by Lillie was found in the rod acromeres of guinea pigs, one rabbit, and several dogs. Treatment with bull testis hyaluronidase (Wyeth) and streptococcus hyaluronidase (Wyeth) for periods up to eighteen hours failed to alter the stainability of the acromeres with thionine, although metachromatic staining of the mucin of umbilical cord matrix and artery was destroyed by the same enzyme preparations within two hours.

Wislocki and Sidman obtained fairly strong metachromasia in the outer segments of the rods in sections of monkey retina fixed in 4 per cent basic lead acetate and stained with 0.5 per cent aqueous toluidine blue. When Orth's fixative was used in an effort to preserve histologic detail more effectively, delicate metachromasia was observed between the outer segments of the rods

and cones. Likewise, with methylene blue, the metachromatic substance appeared to be between, rather than within, the outer segments of the rod. These observations suggested the presence of ground substance or secretion between the photoreceptor cells. In a subsequent paper these same investigators[14] referred to the material between the outer segments and photoreceptor cells and the pigment epithelium as a "readily soluble amorphous substance which exhibits faint and inconstant metachromasia."

Neither Lillie nor Wislocki and Sidman identified the material responsible for the metachromasia about the outer segments of the rods, although the latter investigators suggested that it might be either a lipid or a polysaccharide of weak acidity, such as the hyaluronate of the vitreous. Lillie's observations and mine would seem to exclude the possibility that hyaluronic acid might be responsible. Whether the AMP which I have just demonstrated to you in formalin-fixed human retinas is the same as that responsible for the metachromatic staining of animal acromeres cannot be decided at this time, although I believe it is. Why it seemed to Sidman and Wislocki to be such an elusive soluble substance, yet is so readily demonstrated by the methods I have used, is also not explained. The lack of complete parallelism between metachromatic staining of tissues and their affinity for colloidal iron has been described before. Grishman,[4] for example, found only small areas of metachromasia in the vitreous while much larger accumulations of hyaluronidase-sensitive AMP could be demonstrated with colloidal iron.

For the present, I conclude that in the human retina there is a relatively stable, insoluble ground substance, apparently an acid mucopolysaccharide, in which the rods and cones are suspended. The source, purpose, and precise chemical nature of this material remain to be determined.

PATHOLOGY OF THE RETINA AND OPTIC NERVE

The use of special stains for AMP in the course of our routine histopathologic studies of enucleated eyes has made possible a number of interesting, although not altogether unexpected, observations which relate to alterations in the retina and optic nerve.

1. *Degeneration of the rod and cone layer* is attended by loss of AMP. Depending upon the nature of the degenerative process, there may be an abrupt or a gradual depletion of AMP from one field to the next. On the other hand the entire rod and cone layer may show a loss of AMP, as in the case of total retinal detachment, when pools of AMP may be found scattered throughout the otherwise serous (PAS-positive) subretinal exudate. In this situation a combination of alcian blue with PAS yields striking results. The scattered accumulations of AMP stain an intense blue, while the serous exudate in which they are dispersed provides a vividly contrasting magenta background. More often than not, histiocytes will have made their appearance and ingested the liberated AMP. Under these circumstances the AMP is dispersed within the cytoplasm of the macrophages as fine bluish droplets, but again the contrast with the PAS-positive background is striking.

2. *Microcystic degeneration of the peripheral retina and of the pars ciliaris retinae* is characterized by the accumulation within the cysts of a pale-staining AMP which seemingly is easily lost during preparation of the sections. Often the mucoid material about the periphery of the cysts clings to the cyst wall while that in the center of the lumen has disappeared. This AMP is presumed to be hyaluronic acid, for it is readily rendered nonstainable by hyaluronidase and

its staining characteristics resemble those of the AMP of the vitreous. In cysts of the pars plana much smaller degrees of cystoid degeneration are often observed. In fact all degrees of degeneration may be seen from simple intracellular vacuolization, through a stage in which the cells of the nonpigmented ciliary epithelium are ballooned out by their tremendous accumulation of AMP, to cell rupture and cyst formation.

3. *Retinoblastoma* is a tumor composed, at least in part, of undifferentiated or poorly differentiated neuroepithelial cells. Often in areas where well differentiated Flexner-Wintersteiner rosettes are numerous the impression that these tumor cells are trying to form visual cells cannot be denied. Recalling that the outer ends of well developed adult visual cells are bathed in a mucoid ground substance, it is not surprising to find that the retinoblastoma rosette often contains a similar AMP which also resists the action of hyaluronidase. In areas where the tumor cells remain undifferentiated there may be an intercellular matrix of AMP which is similarly hyaluronidase-resistant.

4. *Cavernous atrophy of the optic nerve*, as seen in sections stained by any of the conventional methods, is characterized by the presence of many large, apparently empty, spaces within the substance of the nerve. The nerve fibers have disappeared in the involved areas, but there has been no replacement gliosis or mesenchymal proliferation as observed in other forms of optic atrophy. Special stains for AMP reveal these cavernous spaces to be filled with hyaluronidase-sensitive AMP. After treatment with hyaluronidase, only the glial framework remains in the areas of cavernous atrophy. Similar mucoid degeneration may be observed within the nerve fiber bundles of the optic disc during the formation of a glaucomatous cup.

5. *Gliomas of the optic nerve* often contain areas of myxomatous degeneration. Such areas may also be seen within the optic nerve at the advancing margin of the tumor. Special stains for AMP give strongly positive reactions in these areas of mucoid degeneration. Treatment with hyaluronidase for periods up to 3 hours fails to affect this AMP-positivity.

SUMMARY

1. Some of the newer techniques for the histologic localization of acid mucopolysaccharides (AMP), based on the affinity of these mucoids for alcian blue and for colloidal iron, have been employed in conjunction with routine histopathologic studies of enucleated eyes.

2. In the normal retina only the layer of rods and cones consistently reveals the presence of significant amounts of AMP. The mucoid here resists hyaluronidase and seems to form a ground substance in which the outer segments of the visual cells are embedded. The normal optic nerve does not yield positive reactions for AMP.

3. Degeneration of the visual cells is attended by a loss of AMP.

4. Microcystoid degeneration of the peripheral retina and of the pars plana is characterized by the accumulation of hyaluronidase-sensitive AMP within the cysts.

5. A matrix of hyaluronidase-resistant AMP is present about some of the undifferentiated cells in retinoblastomas and within the lumens of some of the well developed Flexner-Wintersteiner rosettes.

6. Cavernous optic atrophy is characterized by the massive accumulation of hyaluronidase-sensitive AMP within the cavernous spaces of the degenerated optic nerve.

7. Myxomatous areas within gliomas of the optic nerve contain much AMP which resists hyaluronidase.

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DISCUSSION

GEORGE K. SMELSER, Ph.D., New York: It is usual to express pleasure in the privilege of discussing a paper; this time the cliché can be uttered with particular sincerity because Dr. Zimmerman has written and presented so clearly and with such restraint his work on the application of most modern histochemical techniques to ocular tissues. This makes my task more difficult, for it is much easier to discuss with "criticism-of-interpretation" or to "question the methods."

One aspect of his presentation particularly intrigues me. Dr. Zimmerman has shown that a material, which he suggests may be an acid mucopolysaccharide, exists between the outer segments of the rods and cones in human retina. The important group of tissue substances, which are lumped together under the term of acid mucopolysaccharides, can be demonstrated, as he points out, by four staining methods: (1) periodic acid-Schiff, (2) metachromasia, (3) colloidal iron, and (4) alcian blue. General agreement is developing among histochemists on the limitations of specificity of each of these reactions. Dr. Zimmerman is well aware of this situation. By judicious use of these methods, together with the enzymatic removal of some possible constituents, one can approach the solution of the nature of the substance in which the rod and cone processes lie. He has pressed forward as far

as possible with this line of investigation under the handicaps imposed by staining techniques and his material, i.e., fixed human tissue.

I would be very happy indeed to bring even a minute bit of information to bear on this problem. However, my contribution must take the form of a suggestion, hedged with qualifications. The preparations indicate that the rod and cone processes are embedded in a substance containing acid mucopolysaccharides. By the use of hyaluronidase, the choice of these substances is narrowed to those which are sulfated, or possibly chondroitin. There is one other way of locating the sulfated mucopolysaccharides, at least in animal tissue. If tracer doses of S^{35} are administered, the radioactive sulfate is incorporated in the molecule of mucopolysaccharide. By use of radioautographs the isotopically labelled substance may be located.

This technique, as all others, possesses limitations, this time one not of specificity, but of resolution. Such autographs have been prepared of rabbit retinas, and with due consideration of the difficulty in locating the sulfate with a high degree of precision, one must report that there is no evidence that sulfate-containing compounds are located in the rod outer segments. This indicates that the alcian blue is staining something other than sulfated compounds.

With what does this leave us? (1) Dr. Zimmerman has shown that the rod outer segments are embedded in a matrix of a probably highly polymerized material containing strong acidic groups. (2) This substance may be an acid mucopolysaccharide which is not split by hyaluronidase, but no evidence is available that this substance contains sulfate groups.

What is the importance of this substance, other than that it is there? It is a ground substance of which we were heretofore unaware. It is presumably the medium through which substances necessary for the visual cycle itself are transported. The findings which Dr. Zimmerman has just presented brings to mind the beautiful studies of Kolmer [Arch. f. d. ges. Physiol., 129:35-45, 1909]. Kolmer described droplets of lipid or lipoproteins on the surface and between the rod outer segments. These droplets were identified by the late Professor Detwiler as a vehicle containing retinine, one of the three major points of the rhodopsin-retinine-vitamin A cycle. A decrease of polymerization and, therefore, the density of the alcian blue-positive inter-rod material may well affect the rate of interchange of visual pigment precursors between the pigment epithelium and the outer segments. We, therefore, may not lightly dismiss the report of stainable substances in this strategic location. It is important to learn whether the variation in staining seen by Dr. Zimmerman can be correlated with retinal function in either experimental or diseased eyes.

JOHN E. HARRIS, M.D., Portland, Ore.: Dr. Zimmerman has applied some new techniques to the study of the normal and pathologic ocular structures. I think a discusser should mention certain aspects of the techniques which the author cannot take the time to do while he is giving the paper.

He had applied two techniques. The first involves the deposition of colloidal iron which is subsequently made microscopically visible by applying the classic Prussian blue reaction, and the second, assimilation of a phthalocyanine dye, alcian blue. While exact knowledge of the particular molecular structure identified by these techniques is not certain it can be assumed that under the conditions which Dr. Zimmerman has employed these compounds are polyanionic macromolecules. Among these are the acid mucopolysaccharides. Other possibilities exist. Polyphosphates, for example, must always be considered. However, these are labile compounds and it would seem unlikely that they are involved here.

The alcian blue assimilation, as Dr. Zimmerman has indicated, is probably a more specific test for acid mucopolysaccharides than the deposition of colloidal iron. Alcian blue and metachromasia seem to identify similar groups. However, even assuming that these reactions clearly identify acid mucopolysaccharides, one still has a long way to go. The range of such structures which will give this reaction is broad and it seems likely that fairly subtle changes in structure which appear the same under the microscope may profoundly alter normal physiology.

What can we look forward to? In the first place, as Dr. Zimmerman has reported today, we may be able to demonstrate this general class of substances in normal tissues where we had not expected to find them. Among these, of course, is the material between the outer segments of the rods and cones. With the reservations noted above, there can be no fast issue with Dr. Zimmerman's conclusion that this is an acid mucopolysaccharide.

As to its functions, I would agree with Dr. Smelser that it in some way is related to the nutrition of the rods and cones. Poly-

saccharides in the eye seem to have this key job. For example, the polysaccharide capsule of the lens and the polysaccharide Descemet's membrane are probably important respectively to the nutrition of the lens and to the exchange of fluid which occurs across the endothelial surface of the cornea.

The second technique of Dr. Zimmerman was applied to studies of diseased tissues. In these studies two major alternatives must ultimately be distinguished. The first is that the material taking the stain accumulates secondarily to the disease process and is not etiologically related to the disease. Dr. Zimmerman found an accumulation of positive staining material in the optic nerve, for example, in cavernous atrophy. I believe that this probably represents an accumulation of material which is not etiologically related to the disease. It may be hyaluronic acid and indeed may have come from the vitreous.

The second alternative is most intriguing: accumulation of this material may be etiologically related to the disease. Such a relationship has been postulated in gargoylism, for example, and seems likely in some other diseases. I have been interested in this particular aspect. I have injected a mucopolysaccharide that is foreign to the eye into various ocular structures. Immediately following injection into vitreous, no particular reaction is noted but after a period of time definite changes occur. First, the vitreous becomes liquefied. After a week or more, the cornea becomes edematous and opaque. Still later the eye may become enlarged. There may or may not be an associated increase in intraocular pressure. If the condition persists, the lens becomes dislocated, and may be opaque. Thus, this technique develops a cataract and treats it at the same time. These changes represent a disease process, the cause of which must be the foreign mucopolysaccharide. By extension then, the primary biochemical fault in many diseases may rest in the metabolism of the acid (or neutral) mucopolysaccharides.

Unfortunately the stains employed here are not specific for the foreign mucopolysaccharides. They have the same staining reactions as the normally occurring compounds. Thus, except to demonstrate the presence or absence of a spectrum of molecules, these particular techniques will always be limited. Perhaps a partial answer to this problem will lie in microspectrophotometry, because it seems that, particularly with metachromasia, different compounds have different absorption spectra, albeit appearing much the same under the microscope.

I congratulate Dr. Zimmerman for this excellent paper and I know we all look forward to his future studies.

DR. ZIMMERMAN: When demonstrating these various mucopolysaccharides in ocular tissues we must remember three questions: (1) why are they there? (What is their function, their physiology?); (2) how did they get there? (What produced them?); (3) and perhaps I should have put this first, Just what are they chemically and why are they not all exactly the same? (Witness the differences in response to hyaluronidase.) These are three avenues for future work.

DIABETIC NEUROPATHY AS A CAUSE OF EXTRAOCULAR MUSCLE PALSY

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DIABETIC neuropathy is a common but little understood complication of diabetes mellitus. It occurs most frequently in diabetes of long standing which is not well controlled, and it may involve any sensory or motor nerve. It has been ascribed to the vascular changes commonly found in diabetes, or to vitamin B deficiency, or to the disturbed metabolism which is characteristic of uncontrolled diabetes.[5] This neuropathy is of particular interest to ophthalmologists, since it may involve the optic nerve or any of the motor nerves to the extraocular muscles.

Diabetic neuropathy involving the third, fourth or sixth cranial nerve has been known for many years, but there has been scant mention of the condition in ophthalmic literature. Since life expectancy of the population as a whole is constantly increasing and since treatment of diabetes is constantly improving, more people are living to acquire diabetes and more diabetic patients are living longer. As a consequence, it is not surprising that there is an absolute numerical increase in the number of complications encountered.

Ogle[10] was the first to report paralysis of one of the extraocular muscles complicating diabetes when he cited a case of paralysis of the third nerve. However, careful review of his report casts doubt on whether this was a neuropathy of the kind under discussion. Groenouw[6] collected 647 cases of diabetes from the literature and found that 5.3 per cent of the patients had paralysis of one or more of the extraocular muscles. Köllner[8] approached the problem differently and reported 3 out of 233 cases of ocular muscle paralysis which were the result of diabetic neuropathy. Waite and Beetham,[12] in an outstanding analysis of ocular complications of diabetes mellitus, found only 16 instances of extraocular muscle paralysis in 4001 eyes, and incidence of 0.4 per cent. Leopold[9] studied 100 cases of diabetes mellitus followed for ten or more years and reported an incidence of ocular palsies of 5 per cent of all the cases; he did not feel that this exceeded the incidence of such palsies in the population at large. Collier[2] personally observed more than 30 cases and described the clinical picture. Duke-Elder[4] briefly mentioned that the neuropathy associated with diabetes may be a cause of extraocular muscle paralysis; he stated that the pathologic changes are undetermined and that there may be hemorrhagic or thrombotic lesions. Walsh[13] also briefly mentioned this complication and stated that in his experience it had been quite rare.

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CASE REPORTS

Case 1—Mrs. K. B., aged 53, a known diabetic since 1937, was first seen by me in 1954, at which time she had a complete left external third nerve paralysis with a complete left ptosis and typical outward and downward deviation of the left eye. In retrospect, her case was of particular interest because she had been referred by an internist and a neurologist who had studied her exhaustively, doing, among other things, cerebral arteriography. I was also puzzled regarding the cause of her third nerve paralysis until the late Doc-

N. K. Lazaar suggested that it might be due to a diabetic neuropathy. The patient's blood sugar at this time was 285 milligrams per hundred cubic centimeters. Without other treatment than temporary control of the diabetes, the patient's signs and symptoms cleared up completely over a five-month period. When last seen, two and a half years after all symptoms had disappeared, this patient had remained well.

Case 2—Mr. A. M., aged 47, complained of diplopia which gradually became more annoying over a three-day period. Diplopia fields and objective signs were characteristic of a paresis of the right superior oblique muscle. Examinations revealed a blood sugar of 176 milligrams per hundred cubic centimeters and the presence of urinary sugar, findings which had never been present previously. The diabetes was easily controlled by diet alone, and all diplopia and objective signs disappeared over a three-week period. The patient reported one mild recurrence of the diplopia following an excessive drinking bout, but this could not be confirmed because he was out of the city. This second episode of diplopia promptly disappeared. Two and a half years after the onset and with the diabetes well controlled, there has been no recurrence of symptoms.

Case 3—Mrs. I. M., aged 71, had had diabetes for thirteen years and had been taking insulin for eight years. She experienced a rapid onset of diplopia in 1956, and examination proved the presence of a typical paralysis of the right lateral rectus muscle. In addition she had a moderate diabetic retinopathy. Her blood sugar at that time was 172 milligrams per hundred cubic centimeters. With control of the diabetes the diplopia disappeared over a four-month period, although there remained a residual esophoria of 8 prism diopters for distance and near.

Case 4—Dr. F. O. had a brittle diabetes of many years standing which, in 1956, could be controlled by 50 units of protamine zinc insulin daily. For several months prior to his first visit, he had been traveling a great deal and found it difficult to maintain rigid control of his diabetes. Four days before his first examination he noted the sudden onset of diplopia. At first the picture was confusing, but after four or five days a definite diagnosis could be made of right lateral rectus paralysis. This patient also had a moderate diabetic retinopathy. When his diabetes was again well controlled, his objective and subjective signs and symptoms slowly cleared over a five-month period.

Case 5—Miss M. S., aged 62, first noted vague changes in her vision, with definite diplopia when she was tired, in April 1957. On examination she had an easily elicited diplopia on right lateral gaze, and objective findings of a right lateral rectus paresis. She gave no history of diabetes but her blood sugar was over 200 milligrams per hundred cubic centimeters. Control of the diabetes led to prompt disappearance of her symptoms so that six weeks later no diplopia could be elicited.

Case 6—It is of interest that in reviewing all cases of extraocular muscle paralysis seen in my private practice, a sixth case was discovered which, in retrospect, seems to fit into the category being described, although precise clinical data concerning the diabetes is not available. At the time the patient was seen, any possible connection between the diabetes and the extraocular muscle paralysis was not recognized. Mr. W. J. H., aged 65, was first seen in March 1947, with a history of diplopia of one week's duration. During this week the diplopia had gradually become worse so that on his initial examination there was an obvious right lateral rectus paralysis. The patient had had diabetes for many years but otherwise was in good health. His blood pressure was normal. The diplopia persisted virtually unchanged for about eight weeks, then early in May it rather suddenly and spontaneously disappeared.

DISCUSSION

From these cases and the 30 cases mentioned by Collier,[2] a fairly definite clinical course can be described. Typically, there is a more or less sudden onset of diplopia, the character of which will vary with the muscle or muscles involved. The diplopia may be preceded by vague prodromal symptoms of discomfort or a feeling of fullness. There follows a variable period of bothersome diplopia which appears to depend on the status of the diabetes. If the extraocular paralysis is the initial sign of the diabetes, it is liable to be of short duration. Conversely, if paralysis occurs in a long-standing instance of diabetes which, for one reason or another, is temporarily out of control, signs and symptoms may persist for a period of four to six months.

The outstanding characteristic of this diabetic complication is the fact that it spontaneously improves when the diabetes is controlled or recontrolled. Collier[2] observed that all of his patients recovered completely, although Groenouw[6] observed that in some of the cases which he collected from the literature, paralysis was permanent. It is noteworthy that one of the patients here reported recovered, in spite of the fact that her blood sugar was only temporarily controlled for a short time immediately following onset of diplopia.

In the differential diagnosis, cerebral vascular disease must be considered first. Rucker and his co-workers[10] classified 168 of their 653 cases as being due either to aneurysm or vascular causes, indicating this to be a common etiologic basis. Since most patients over 50 years of age who have diabetes also have a certain amount of vascular damage, it is difficult to exclude this factor. Indeed, in some cases it may be impossible to decide whether a paralysis is on a vascular basis or is due to diabetic neuropathy.

From the standpoint of tissue changes, the differential diagnosis is also difficult. The only description of the pathologic changes of diabetic neuropathy involving the motor nerves of the extraocular muscles was published recently by Dreyfus, Hakim and Adams.[3] These authors reported finding a gross fusiform enlargement of the retro-orbital part of the third nerve in a diabetic patient who had clinical signs of a left third nerve paralysis. This patient died following cerebral arteriography. On section, destruction of some of the myelin sheaths and axis cylinders in the center of the nerve was found. In addition, there was increase in the epineural, perineural and endoneural connective tissue and suggestive evidence of regeneration in the area of destruction. The vasa nervorum showed arteriosclerosis, and the authors felt that the whole picture could be accounted for on the basis of incomplete ischemic neuropathy. They could not, however, find any actual occlusion of a nutrient artery. Woltman and Wilder,[14] in pathologic specimens from the lower extremities, found patchy, irregular degeneration of the myelin sheaths which was invariably associated with occlusive vascular phenomena of greater or lesser degree.

It is difficult to reconcile all the facts of the clinical aspects of these cases with the pathologic picture. Many questions remain unanswered. The relationship between the disturbed metabolic process and the vascular changes in the pathogenesis of the neuropathy is uncertain. The reason why some diabetic patients with arteriosclerosis develop the neuropathy while others do not remains a mystery. The fact that the neuropathy may be the initial symptom, as in case 2 and case 5, or may occur in a patient with long-standing diabetes which is out of control is hard to explain. The occasional spontaneous recovery from the neuropathy without complete control of the blood sugar, as occurred in case 1 and as described by Jordan,[7] also is unexplained.

Whatever the answers to these questions, the fact remains that sound clinical evidence strongly suggests a direct connection between the diabetes and the neuropathy. Breinin[1] has recently pointed out that electromyography shows unique changes in these cases, further confirming the fact that there is such a connection. This relationship is such that all patients suffering sudden paralysis of those muscles supplied by the third, fourth or sixth cranial nerve should be examined for possible diabetes.

SUMMARY AND CONCLUSIONS

Six cases of extraocular muscle paralysis apparently due to neuropathy of diabetes mellitus are reported.

The increase in incidence of diabetes and the longer life expectancy of diabetic patients suggest that these cases may be more common than has previously been thought, and consequently the existence of diabetes mellitus should be excluded in every case of paralysis of the extraocular muscles in which the cause is not obvious.

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DISCUSSION

IRVIN H. LEOPOLD, M.D., Philadelphia: Dr. Snydacker has directed our attention to the occurrence of extraocular muscle palsies as a complication of diabetes mellitus. He noted that this has been mentioned infrequently in the ophthalmic literature. It is important for the ophthalmologist to be aware of the possibility of this condition and that it is not an extremely rare cause of ocular nerve palsy for, as Dr. Snydacker has pointed out, palsies of this origin usually carry a favorable prognosis.

Within recent years, a number of reports of this complication of diabetes have been made. Most of the authors have been impressed with the headache that accompanies the extraocular nerve palsy. There was one individual reported by Hirson in 1948 (Hirson, C., Proc. Roy. Soc. Med., 41:104, 1948) and in 1955 Jackson described four more cases of ocular nerve palsy associated with severe headache occurring in diabetic patients (Jackson, W. P. U., Brit. M. J., 2:408, 1955). Waind recorded two more patients in 1956 (Waind, A. P. B., Brit. M. J., April 21, 1956) and Lincoff and Cogan added four in 1957 (Lincoff, H. A., and Cogan, D. G., A.M.A. Arch. Ophth., 57:181, 1957).

An outstanding feature of this complication is that recovery usually takes place within a few months and may do so, as in a patient of Waind's and in one of Dr. Snydacker's, independently of treatment. Similar cases have been found to occur in individuals who showed no evidence of diabetes and in whom recovery also occurred spontaneously. This may suggest that the cause of the diabetic and nondiabetic extraocular involvement in those which improve is the same and that therefore the syndrome is not specific to diabetes, although it may well be relatively more common in that condition.

The symptom combination of unilateral frontal headache and oculomotor paralysis has long been recognized by clinicians as characteristic of aneurysm of the intracranial portion of the carotid artery. Dr. Snydacker, as

well as Eareckson and Miller, Waite and Beetham, Walsh, Waind, Jackson, and Lincoff and Cogan have shown that the same group of symptoms can be found in patients with diabetes who do not show evidence by arteriography and subsequent clinical course of intracranial aneurysm of the carotid artery. Recovery can occur in cases due to aneurysm, and the time of recovery may vary from a week to a year, or longer. Some, of course, do not recover completely and others, not at all. Of course one may find other signs of aneurysm, such as stiffness of the neck or blood in the spinal fluid, which would be strong evidence against the association with diabetes and in favor of aneurysm. However, if one has a patient with diabetes who presents this classical aneurysm syndrome, an arteriogram would be helpful but, if the patient is over 60 years of age with diabetic retinopathy and negative spinal fluid, in the absence of signs of meningismus, one would be justified in postponing carotid arteriography.

It is well to keep in mind that other conditions will produce the same group of symptoms. Tumors must be considered, but these usually may be diagnosed by changes in roentgenograms. Lincoff and Cogan have also collected cases which have been due to leukemia and to ophthalmoplegic migraine. The leukemia case could be diagnosed by the evidence of lymphocytosis in the spinal fluid and the other signs of leukemia. Patients with ophthalmoplegic migraine give a history of recurrent attacks of the syndrome with quick and full recovery. In none of these other cases were there evidences of stiff neck or bloody spinal fluid. The majority of Dr. Snydacker's patients were over 50 years of age. Headache was not an outstanding symptom in his series, so one must be aware of this complication in diabetic patients in the older age group whether or not headache is associated.

Speculation has been made concerning the etiologic basis and the exact nature of the lesion. It is suggested that it is of vascular origin, perhaps a minute hemorrhage either into the nerve trunk or about the nucleus of the oculomotor nerve. This theory is supported by the rapidity of onset in a great majority of cases with complete paralysis of the nerves involved. The tendency toward total regression has been mentioned, although recovery of function may require several months. Vitamin deficiencies frequently noted in diabetes mellitus and polyneuritis have also been suggested as causes of the ocular palsies. A recent pathohistologic observation has been made in diabetic oculomotor palsy by Adams, Hakim and Dreyfuss. They noted a gross swelling of the intraorbital portion of the third nerve with extensive destruction of medullated fibers in the central portions of the nerve.

An awareness of this syndrome and the exclusion of the other possible causes in a patient with diabetes mellitus allows one to give a more favorable prognosis. The exact frequency of this complication has not been established firmly, and further work is required to determine the exact pathogenesis of these pareses.

DR. SNYDACKER: I would like to thank Dr. Leopold for his very kind discussion. He has pointed out that we should be constantly aware of the fact that there are many possible causes for extraocular muscle paralyses. If a patient is seen with an extraocular muscle paralysis of a relatively sudden onset, that patient should be referred to his internist and to a neurologist. If no cause for the extraocular muscle paralysis can be found, and if there is a proved diabetes mellitus, I believe that we are justified in assuming that the patient has a diabetic neuropathy. Rigid control of the diabetes should be carried out without other treatment for a reasonable period of time.

It is important that these patients be followed clinically both by the internist and by the neurologist, but, as Dr. Leopold has said, it can be suggested that the prognosis is good. This is exactly the procedure that I followed in the case of the physician who was reported in this series. Although I had reasonable faith that I was correct, when his severe, bothersome diplopia lasted for four months, I began to be just a little concerned, although I tried not to show it to him. Eventually after five months, my faith was justified and his diplopia disappeared.

DACRYOCYSTORHINOSTOMY

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THE surgical management of chronic dacryocystitis is indicated in those cases of the disease which are resistant to local measures and antibiotic therapy. Most patients in this category will be found to have an obstruction in the lower portion of the lacrimal sac or in the nasolacrimal duct. Pathologic nasal conditions, such as deviated septum, polyps and chronic sinusitis, must be excluded as possible causes of lacrimal obstruction before surgical intervention is used. In any event, they must be adequately treated.

Patients with chronic dacryocystitis ordinarily suffer from chronic lacrimation, sometimes accompanied by a purulent discharge from the lacrimal sac and by chronic conjunctivitis. Dacryocystectomy, which formerly was the favored procedure, has the basic defect that, although it removes the site of the infection, it does not correct the lacrimation, which continues because the tears still have no way to get into the nose. It has been almost entirely replaced by dacryocystorhinostomy, an operation which establishes a new connection between the lacrimal sac and the nasal cavity and thus answers the objection to dacryocystectomy. Although otolaryngologists have attacked the problem via the nasal route in their competition with ophthalmologists for the management of this condition, most operations done today to re-establish lacrimonasal drainage are done through the external approach.

From the Section of Ophthalmology, University of Puerto Rico School of Medicine.
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External dacryocystorhinostomy was first described by Toti,[8] in 1904. The technique has since been modified by many surgeons,[1,3-6] notably Mosher,[7] and at the present time, ophthalmologists differ widely in the technique they employ. One of the most useful recent advances is the use of the Stryker saw in opening the lateral bony wall of the nose.[6] This instrument simplifies the technique, shortens the operating time, and eliminates the risk of damage to the soft tissues by its rapid oscillating action, in contrast to the trauma to these tissues which frequently occurs when dental or other burrs, bone chisels or rongeurs are employed.

TECHNIQUE

The following technique of external dacryocystorhinostomy has been found simple, safe and uncomplicated. The almost complete absence of bleeding is one of its most desirable features. Several of the steps of this operation were originally described by Dr. Ramón Castroviejo.[2]

Anesthesia

The best results are obtained with general anesthesia, re-enforced with local anesthesia applied to the nasal mucosa and the superficial tissues over the lacrimal sac. A cotton tampon, well moistened with 4 per cent cocaine solution and Adrenalin (1:1,000) is introduced into the nose and is placed in contact with the area of nasal mucosa which corresponds to the lacrimal fossa. The resulting shrinkage and ischemia prevents bleeding when the mucosa is incised. A piece of thread attached to the tampon facilitates its removal at the ap-

propriate stage of the procedure. Three cubic centimeters of Novocaine solution, to which 2 drops of Adrenalin (1:1,000) have been added, are injected into the superficial tissues over the lacrimal sac.

Incision

A vertical, slightly curved incision, 3.5 cm. long, is made 3 or 4 mm. nasal to the internal canthus through the skin and subcutaneous tissues up to the internal canthal ligament. The Stevenson lacrimal sac speculum is then introduced.

Procedure

The internal canthal ligament is cut with scissors close to its insertion at the anterior lacrimal crest, after which the overlying periosteum is incised with a knife and is separated anteriorly with a periosteal elevator, leaving the bone bare. With this technique there is no bleeding because the angular blood vessels which pass just anterior to the lacrimal crest are not injured.

The lacrimal sac, which is located in the lacrimal fossa immediately posterior to the canthal ligament, is dissected free laterally from the lacrimal fossa. The periosteum is also separated from the lacrimal fossa.

A lacrimal probe, with an opening at the tip, is passed through the lower canaliculus, into the lacrimal sac.

A vertical incision is then made in the nasal aspect of the lacrimal sac with a knife and scissors. A needle threaded with 2-0 silk is passed through the hole in the probe, which has been exposed by the incision. One end of the suture is brought out through the canaliculus and punctum lacrimale by withdrawing the probe.

An opening is made in the nasal bony wall with the 10 mm. Iliff trephine of the Stryker saw. A flap of nasal mucosa is dissected along the border of the bony opening but is left attached posteriorly. This flap is sutured to the posterior flap of the incised lacrimal sac with three sutures of 3-0 chromic catgut.

The cotton tampon is now removed from the nose and the other end of the silk suture in the lacrimal sac is brought down outside the nose, where it is tied to the end already brought out through the punctum.

Closure

The anterior flap of the incised lacrimal sac is sutured to the periosteum which was retracted anterior to the bony opening with three sutures of 3-0 chromic catgut. The skin is closed with a continuous 6-0 silk suture. A light dressing is applied.

Postoperative Management

The silk suture in the new nasolacrimal tract is removed from it on or about the seventh postoperative day. The skin sutures are removed at the same time. The lacrimal passage is irrigated at the end of the second week after operation and at weekly intervals thereafter for about two months.

SUMMARY

Chronic dacryocystitis resistant to local measures and antibiotic therapy requires surgical treatment. Dacryocystectomy, formerly the favored procedure, does not correct lacrimation. Dacryocystorhinostomy meets this objection, and, when performed by the technique described, is a simple, safe and practically bloodless operation.

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A CASE OF BEHÇET'S DISEASE

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THE histologic examination of an eye affected with Behçet's disease has only rarely been reported in this country. We had occasion to examine such an eye which, in addition, showed some unusual features not previously recorded.

A 46-year-old white man was first seen in August 1954. At that time he complained about pain in the left eye which had persisted for ten to eleven months. This pain came in attacks during which the eye was red and painful. These attacks lasted two to three days and occurred nearly once every month.

When first seen the patient had a hypopyon iritis in the left eye. He was treated locally with atropine and heat. Systemically he received fever therapy. In order to clear up any possible focus of infection, all teeth were extracted and a Caldwell-Luc operation was done on the right side because of an infected maxillary sinus. The condition improved temporarily, but the hypopyon recurred in September. The vision slowly dropped from 6/9 to 6/30. Another attack occurred in November 1954. Results of all laboratory tests remained negative.

The patient returned in October 1955 with another attack of hypopyon iritis on the left side. The eye was now blind. For the first time he complained about a sore in the mouth. He had afterwards repeated attacks of aphthous stomatitis. He was seen by the dermatology clinic and they concurred in the diagnosis of Behçet's disease. Genital lesions were never found.

A paracentesis was done on October 7, 1955. Bacteriologic and virologic examination of the purulent fluid resulted in only negative findings. Intensive steroid treatment was started. The eye became more painful and was finally enucleated on January 10, 1956.

On histologic examination of the enucleated eye (no. 2957), an oblique scar could be seen in the cornea. The anterior chamber was filled with fibrin erythrocytes and organized blood.

Foci of round cell infiltration could be found in the iris (fig. 1), ciliary body, anterior choroid (fig. 2), retina, episclera and within one extraocular muscle present on the sections. These foci consisted of lymphocytes, plasma cells and large cells with a vesicular nucleus, presumably reticulum cells (fig. 3). The retina was completely detached and a large hemorrhage was present in the epichoroidal space (fig. 2). The lens capsule was ruptured anteriorly and marked cataractous changes were present in that area.

This case of Behçet's disease is, therefore, characterized by a widespread inflammation not only in the uvea and the retina, but also extraocularly in the episclera and in the extraocular muscles. The inflammation is predominantly perivascular, as described in previous examinations.[1-3] Unusual is the occurrence of many reticulum cells or immature lymphocytes in these foci. The iatrogenic rupture of the lens capsule occurred three months before the enuclea-

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FIG. 1—Round cell focus in the iris.

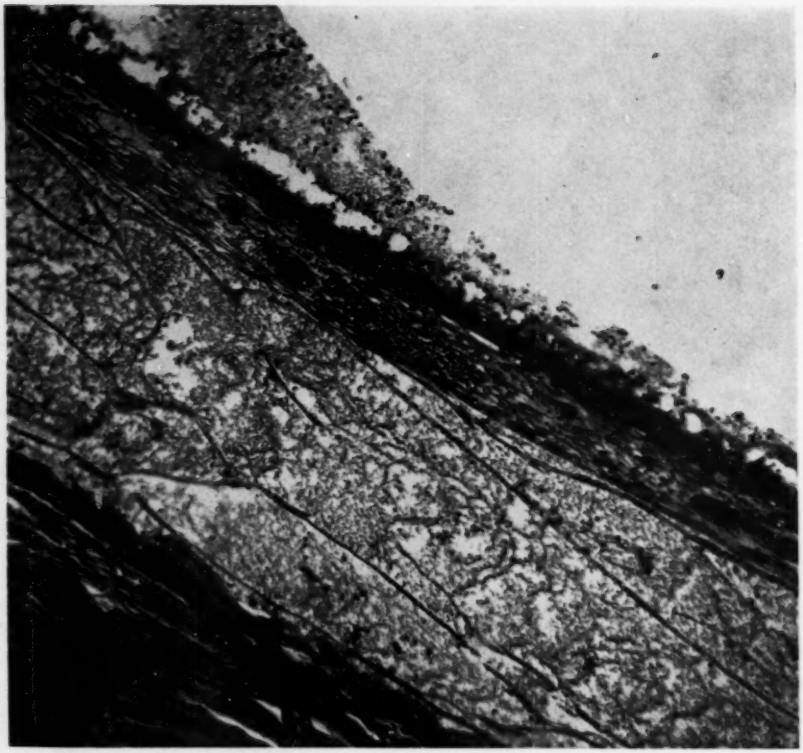

FIG. 2—Round cell focus in the anterior choroid. There is a large hemorrhage in the epichoroidal space.

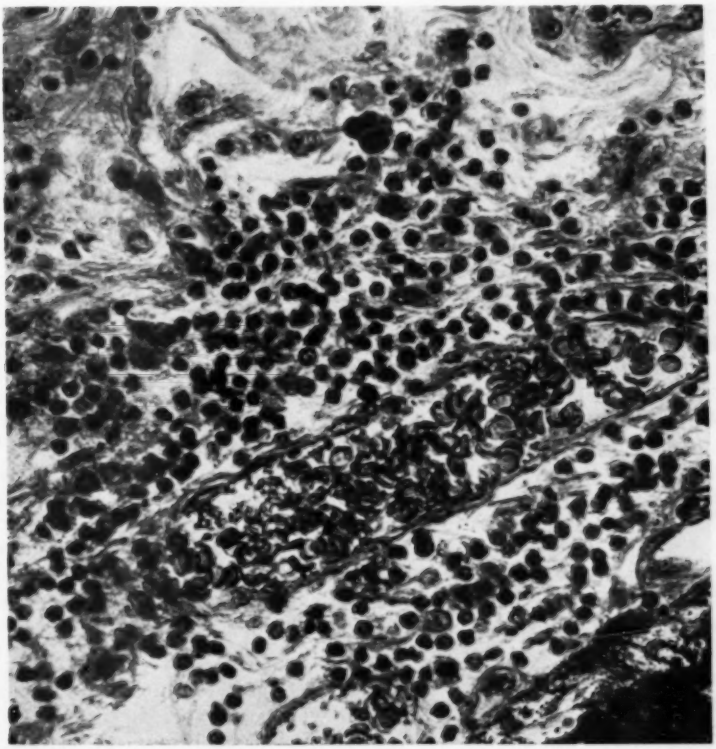

FIG. 3—One inflammatory focus in higher magnification. There is marked perivascular infiltration. Large round cells can be seen in the periphery.

tion and apparently did not influence the original inflammation.

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COGAN'S SYNDROME

Case Report and a Review of Etiologic Concepts

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AN INCREASING number of cases of a perplexing combination of symptoms have been reported in the past twelve years. This symptom complex consists essentially in the association of nonsyphilitic interstitial keratitis with vestibuloauditory dysfunction, and it has acquired the title *Cogan's syndrome*. The cause is unknown and treatment to date has not been consistently satisfactory.

The syndrome usually occurs in young adults and is of rather sudden onset. The initial ocular symptoms include redness of the eye, blurred vision, pain, lacrimation and blepharospasm. The vestibuloauditory manifestations are vertigo, nausea, vomiting and diminished hearing, closely resembling the symptoms of Ménière's disease. These manifestations diminish with the onset of deafness, which often progresses to severe and permanent hearing loss.

Biomicroscopic examination of the eye demonstrates a grayish, granular corneal infiltrate found predominantly in the deeper layers of the peripheral cornea. There may be little or no reaction in the anterior chamber or iris. Late in the course of the disease, interstitial vascularization of the cornea develops. The neurologic examination is usually normal. Tests of cochlear function indicate a profound and often complete loss of auditory perception, while response to labyrinthine stimulation by rotation or caloric methods is entirely lacking. Leukocytosis is present and is often associated with mild eosinophilia. The syndrome usually runs a chronic course, with multiple unpredictable recurrences. The vestibuloauditory deficit, however, once established, tends to persist indefinitely. This is particularly true of the labyrinthine responses.

A case such as this has recently been studied at the University Hospital of the University of Michigan. This case is noteworthy because of the clinically established diagnosis of periarteritis nodosa and also because it showed a perilimbal superficial keratitis which progressed to interstitial vascularization of the cornea.

REVIEW OF LITERATURE

The earliest description of coexisting vestibuloauditory disturbance and nonsyphilitic interstitial keratitis was published by Mogan and Baumgartner[11] in 1934. In the case they described, the auditory symptoms were relieved by cervical ganglionectomy. The history of this symptom complex as a separate entity, however, began in 1945, when Cogan[2] described four patients with interstitial keratitis associated with vertigo, tinnitus and profound perceptive deaf-

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ness. Despite a careful review of his cases, Cogan was unable to ascertain the etiologic agent. He did emphasize the absence of syphilis, but at that date this disease could not be unequivocally excluded. Rosen[14] in 1949 reported a case of interstitial keratitis and vestibuloauditory symptoms following vaccination for smallpox. It was his impression that the syndrome might be attributed to an antigen-antibody reaction in a hypersensitive individual.

Cogan[3] in 1949 described four additional cases of the syndrome and pointed out that in these cases at least, the vestibuloauditory symptoms came on within a few days of the ocular symptoms. All of his patients were young adults. He again emphasized that the only laboratory abnormalities were a somewhat elevated white blood cell count, eosinophilia, and increased sedimentation rate. These findings were the only evidence of systemic disease. Robson[13] in 1950 reported the syndrome in a 27-year-old man and emphasized that all treatment was ineffective, including a bilateral cervicothoracic sympathectomy. Donald and Gardner[4] in 1950 reported the appearance of the symptom complex in a 35-year-old man and emphasized their inability to establish a cause for the disease, indicating that superior cervical ganglionectomy had not helped materially.

Lindsay and Zuidema[9] in 1950 discussed three cases and stressed the significance of viral infection as a possible causative agent. In 1952 Hedges and Taylor[5] described the association of vestibuloauditory symptoms and uveal tract disease in a 20-year-old Negress with proved Boeck's sarcoid. Oliner and others[12] in 1953 gave an excellent review of the information gathered on this symptom complex up to that date and reported one case in great detail. This case was unique in that generalized essential polyangiitis developed after two episodes of vestibuloauditory and ocular symptoms, suggesting a relationship between the two entities. Boyd[1] in 1957 reported two cases of Cogan's syndrome in which the associated clinical findings strongly suggested periarteritis nodosa.

REPORT OF A CASE

The patient was a 60-year-old white woman who first came to the University Hospital on November 14, 1956, referred by her local osteopathic physician with a diagnosis of septicemia due to *Staphylococcus albus*. She had been in good health until March 1956, when she developed a severe upper respiratory infection accompanied by redness, watering, and soreness of the eyes which did not improve under local treatment. Approximately two months after the onset of symptoms she noticed that her vision had begun to fail, and a month later she became suddenly deaf in the right ear. Within three weeks the left ear had become totally deaf also. The hearing loss was accompanied by a staggering gait, subjective unsteadiness, and vertigo. From the inception of her illness she had noted severe intermittent frontal and temporal headaches, with migratory and generalized pains throughout her body and persistent lassitude and fatigue.

Her past medical history included a tonsillectomy in 1946 and an appendectomy in 1950. She had been treated with injections "for her blood" during the past twenty years and had noticed swelling of the ankles for many years. There was no history of arthritis. One brother had developed hearing loss late in life but family history was otherwise noncontributory.

When the patient was first seen in this hospital she was totally deaf and there was no response to maximal caloric stimulation of the vestibular labyrinths. Visual acuity was finger counting

bilaterally. There was a marked circumcorneal and episcleral injection of both eyes. Photophobia and tenderness were prominent. Saccular dilation in association with the sludging of the red blood cells was noted in the immediate limbal conjunctival vessels. The iris and lens were normal. On the right disc there was a linear hemorrhage which appeared superficial. The macular area and retinal periphery were normal bilaterally. Two small petechiae were noticed on the right buccal mucosa. A small thyroid nodule and a grade one systolic apical heart murmur were noted. There was marked thinning of scalp and body hair. Her temperature ranged intermittently up to 102.4 F. but dropped precipitously to normal and remained there upon commencement of steroid therapy. The blood pressure was 120/70 and the pulse was 94.

Admission laboratory studies revealed a hemoglobin level of 10.7 grams per cent, hematocrit of 37 per cent, and a sedimentation rate of 32 mm. per hour. The white blood cell count was 15,450, with a differential count of 7 banded and 79 segmented polymorphonuclear cells, 7 small and 5 large lymphocytes, 2 monocytes, and no eosinophils. The white blood cell count performed on November 29, 1956, totaled 20,200; the differential count showed 2 banded and 76 segmented polymorphonuclears, 8 small and 10 large lymphocytes, 2 monocytes, and 2 eosinophils. Results of urinalysis were within normal limits. The hemoglobin level declined to 8.5 grams per cent, requiring a transfusion of 1000 ml. of whole blood. A stool specimen was negative for occult blood. The nonprotein nitrogen level was 29 mg. per cent. The total serum protein level was 5.9 grams per cent with albumin 3.8 and globulin 2.1 grams per cent. The liver test battery was normal. Lupus erythematosus preparations were negative on four occasions. The C-reactive protein level was five plus. The blood Kahn reaction was four plus, but the *Treponema pallidum* immobilization test was negative on two occasions. Blood cultures on nine occasions showed no growth. The cerebrospinal fluid contained a total of 9 cells per cubic millimeter with a total protein level of 41 mg. per cent, a chloride level of 116 mg. per cent, and a sugar level of 26 mg. per cent. The spinal fluid Kahn reaction was negative and the colloidal gold curve was flat. A spinal fluid culture was negative. A 24-hour urine test for arsenic showed none of this element to be present. A tuberculin test in 1:100 dilution showed a 1 cm. induration. Roentgenograms of the skull and chest revealed no abnormalities and roentgenograms of the pelvis showed no evidence of any heavy metal injections. Biopsy examination of the skin and muscle was negative.

The patient was discharged on a regimen including Meticorten 15 mg. every six hours, Isoniazid 100 mg. three times a day, Pyridoxine 25 mg. three times a day, and a reduced salt diet.

The patient was readmitted on January 24, 1957, for further study. At this time visual acuity was 20/100 in the right eye and 20/50 in the left eye. There was a bilateral localized, raised area of hyperemia of the conjunctiva and episcleral tissues, contiguous to the limbus temporally and superiorly. Slit lamp examination revealed a perilimbal ring distribution of questionable lipid deposits (cholesterol?) in the superficial peripheral corneal stroma. Occasional cells were visualized in the aqueous but no flare was seen. The ophthalmoscopic examination was negative bilaterally. Results of the physical examination and vestibuloauditory tests were essentially unchanged. The admission urinalysis was normal. The hemoglobin level was

12.8 grams per cent and the white blood cell count was 7600. The corrected sedimentation rate was 31.6 mm. per hour. Biopsy of the skin and muscle showed no evidence of collagen disease. The blood Kahn reaction was positive with a 1:4 titer. The patient was discharged with instructions to continue therapy with Meticorten 15 mg. total daily, and Neo-Delta-Cortef eye drops.

The patient was examined again on March 14, 1957, complaining only of occasional redness of the eyes, mild and transient vertigo, and continued total deafness bilaterally. The urinalysis showed a specific gravity of 1.021, an albumin reaction of two plus and myriads of red blood cells. The total serum protein level was 7 mg. per cent with an albumin-globulin ratio of 1.8. The serum creatinine level was 0.43 mg. per cent. The C-reactive protein level was five to six plus and the Bromsulfalein retention was 19 per cent in 45 minutes. The blood Kahn reaction was positive in 1:2 dilution, but results of the *Treponema pallidum* immobilization test again were negative. The serum contained a large amount of substance similar to fibrin and formed an agar-like coagulum upon standing at room temperature. The free moving boundary electrophoretic analysis of the serum showed a low albumin fraction, greatly increased $alpha_1$ and $alpha_2$ globulin fractions, and normal beta and gamma fractions.

COMMENT

Etiologic explanations for this disease have not been entirely satisfactory, but the syndrome has been associated with the following circumstances: vaccination,[3,14] recent upper respiratory infection,[2,4] exposure to Lewisite,[2] ptomaine poisoning,[2] periarteritis nodosa,[1,7,12] Boeck's sarcoid,[3] drug sensitivity,[15] generalized lymphadenopathy,[16] and a history of hypersensitivity.

In our case, it is interesting to note that the features of Cogan's syndrome were somewhat atypical in that the keratitis was perilimbal, confined to the superficial one half of the corneal stroma, and associated on several occasions with paralimbal episcleritis. In this respect, the ocular signs and symptoms closely resemble those described by Hogan[6] as associated with atopic eczema (neurodermatitis), an eczematous perilimbal infiltration being the essential feature of the cases he reported. Acne rosacea keratitis and a few cases of phlyctenular keratoconjunctivitis may occasionally exhibit corneal changes resembling those seen in our case. Similar ocular signs and symptoms, furthermore, have been associated with such hypersensitivity states as rheumatoid arthritis, Wegner's granulomatosis, periarteritis nodosa, Reiter's syndrome and Beçhet's syndrome, to mention only a few. Vestibuloauditory disturbances with actual histologic changes in the eighth nerve have been found in proved cases of periarteritis nodosa.[10,12] These symptoms are common to Ménière's disease and are seen in states in which the blood supply to the labyrinth is suddenly interrupted, as in labyrinthine hemorrhage or thrombosis.[8]

It appears that certain features peculiar to Cogan's syndrome may thus be associated with hypersensitivity states, among them periarteritis nodosa. It has been postulated[17] that such hypersensitivity states are the reaction of a dysfunction of the autonomic nervous system to some internal or environmental stress or both (cold, light, heat, bacteria, drugs, and so forth). It has been emphasized that in such affected individuals the altered autonomic dysfunction is thought to be determined by that person's heredity. The specific end organ or tissue response to the causative stress is thus governed by the peculiar

constitutional make-up of the affected individual. Such a constitution, and even the end organ response, may be hereditarily determined. If such is the case, we may eventually anticipate seeing such hypersensitivity states and possibly Cogan's syndrome occurring in several members of the same family.

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DISCUSSION

EDWARD W. D. NORTON, M.D., New York: A year and a half ago, two patients were seen at the New York Hospital-Cornell Medical Center, with the clinical picture of nonsyphilitic interstitial keratitis and vestibuloauditory symptoms. One was a man, aged 29, and the other a woman, aged 31. Both patients had signs of severe systemic disease compatible with the clinical diagnosis of polyarteritis. In fact, these patients were presented to medical grand rounds as patients in the terminal phase of polyarteritis with so-called Cogan's syndrome. However, neither patient had a histologic picture of polyarteritis on skin and muscle biopsy nor did they follow the expected course of patients with acute fulminating polyarteritis. Today, these two patients, who seemed to be at death's door one and a half years ago, appear to have made a complete recovery except for residual tinnitus and deafness and minimal corneal scarring.

At the time we studied the cases, three questions arose:

First, is this an entity that deserves recognition as a syndrome or is it a chance combination of signs in a widespread disease?

Second, what is its relationship to polyarteritis?

Third, what is its causation?

After careful review of the literature, it seems to me that this condition does deserve recognition as a syndrome characterized by more or less simultaneous onset of bilateral tinnitus, vertigo, deafness, and patchy interstitial keratitis with associated leukocytosis in young adults. Unfortunately, this does not advance our understanding of the condition, but it does arouse interest in it and facilitates discussion, if nothing else.

What is its relationship to polyarteritis? From the literature and from our own experience, one is immediately impressed by the similarity of some of these cases to polyarteritis. However, in the majority of the cases biopsies have given negative results; what is more important, the clinical course of the disease has been different from that in the usual cases of polyarteritis. Generally speaking, one would expect patients with such widespread evidence of vascular disease to be dead within several years, but few, if any, of these patients have died of the disease; therefore, I believe the relationship to polyarteritis is far from established, and I do not see what is gained by classifying it as such.

What is its causation? As Dr. Quinn has pointed out, no satisfactory explanation has been established. In view of the eosinophilia which many of the patients show and the histologic picture of fibrinoid collagen alterations along with arteritis that has been seen occasionally, it is natural that thought has been directed toward hypersensitivity states. However, as Klemperer has emphasized, insults other than hypersensitivity can elicit a similar histologic picture. I think it is fair to say that we have much to learn about the etiology of this syndrome.

In view of the apparent recovery of our two patients, it would seem to be a fair question to ask what treatment was used. I can only answer that all concerned with these two cases agree that the patients recovered in spite of us and not because of us. Corticosteroids did not seem to be effective.

I would like to congratulate Drs. Quinn and Falls for bringing this interesting problem up for discussion.

DR. FALLS: Thank you, Dr. Norton. You have added immeasurably to the outline and details of this particular syndrome.

The important thing about any particular disease entity that has been recognized as a syndrome is that as both otologists and ophthalmologists we have one prerogative, that of considering the individual as a whole. It thus behooves us to be aware of the fact that the eye and the ear can definitely present changes that are diagnostic of widespread systemic disease.

In the case we have presented, one of the reasons we have felt that undue immunologic reactions were occurring was the fact that the serum had a tendency to coagulate on standing, and that there was, in addition, a markedly abnormal electrophoretic curve suggesting disturbances in the serum proteins. It was our interpretation that at least a very unusual autonomic response was occurring.

In closing I should like to emphasize, as has Dr. Norton, that one must consider the individual as a whole when encountering unusual otologic or ophthalmic pathologic changes. I would like furthermore to stress that the individual is a product of his heredity and environment. Since there appears to be a great increase in the number of collagenous diseases that we are seeing, it is most important that, as physicians, we inquire into the possibility of allergic reactions either in the individual or his relatives whenever injecting any drug or foreign protein. No foreign protein material should ever be administered intramuscularly or intravenously to anyone without first taking the simple expedient of inquiring into the possibility of previous drug sensitivity.

It has been a distinct pleasure to discuss this paper.

SYMPOSIUM: THE SIGNIFICANCE OF OCCUPATIONAL HEARING LOSS TO THE OTOLARYNGOLOGIST

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MODERATOR NASH: The title of this symposium begs the question. Technically it presents a nondebatable subject. If we had said, "Is occupational hearing loss significant to the otolaryngologist?" it would have been a real issue and it would have been a debatable question. Apparently the originator of this designation felt that a knowledge of occupational hearing loss is significant; as a matter of fact, it is important to the otolaryngologist. I might tell you, so do we.

Years ago Roy Chapman of the Reptile Institute of New York made this statement, "Of all the creatures in the animal kingdom, the King Cobra is the most vicious and dangerous member." Later, a prominent reptilian expert in London, Sir Archibald Squire, took exception to Mr. Chapman's statement, and in support of his negation he cited this instance: One day on a golf course in Australia, a player picked up and handled a King Cobra and nothing happened. Sir Archibald explained that the player thought that the reptile was a python.

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This story of the animal kingdom I should like to compare to a similar situation in the field of industrial noise and occupational hearing loss, for in both we have the situations:

1. A condition of exposure and liability to injury
2. A difference of opinion concerning the danger of such exposure to liability and injury
3. An unscientific approach to study of the problem
4. Conclusions drawn from inadequate, irrelevant and unacceptable data.

By way of history (and you should have a little history of this subject), may I tell you that prior to 1945, workers exposed to damaging noise apparently accepted occupational loss of hearing as a part of their jobs, which were always lucrative ones and which presently pay from $85 to $210 a week, and such pay is good money.

As judicial opinions and interpretations of compensation laws became more generous and as disablements as well as disabilities became compensable, workers recognized that the time had arrived when they could be rewarded for a hearing loss incurred from their jobs; and industry realized that it was being confronted with an almost insurmountable

compensation bill for which it had set aside no financial reserves.

In an attempt to work out the complexities of a situation in which the worker had made claim for a real disablement and for which the manufacturer had not set aside adequate funds to meet the claim, there arose all sorts of problems and all sorts of questions, many of which required immediate answers (even if the answers were wrong), because without these answers adjudicating bodies were not able to process claims, not to mention the awarding or denying of recompense.

For your information may I say that the members of this panel do not know all the answers, but they do know the questions, and in my opinion they have had more experience with the problem of industrial noise and occupational hearing loss than any other group so far as the medical aspects of industrial noise are concerned.

I want to introduce these men to you.

First, Dr. Hallowell Davis, director of research of the Central Institute for the Deaf, St. Louis, and the executive secretary of "Chaba."

Hallowell, what is Chaba?

DR. DAVIS: I had better put it on the blackboard. C-h-a-b-a. Chaba is the Committee on Hearing and Bioacoustics. The Committee on Hearing and Bioacoustics is a Joint Committee of the Armed Forces and the National Research Council to advise the Armed Forces on questions of hearing such as we have been discussing today. Another field is bioacoustics, which includes other effects of sound on man and on animals. Another is the community reaction to airplane noise, and suppression of such noise represents still another.

MODERATOR NASH: That gives us some idea of Chaba.

Dr. Meyer Fox is from Milwaukee. He is a member of the Committee of Medical Consultants to the Industrial Commission of the State of Wisconsin, and the top otologist in occupational hearing loss of that state.

Dr. Aram Glorig of Los Angeles is director of the Research Center of the Subcommittee on Noise in Industry, and he is a former chief of audiology of the U. S. Veterans Bureau in Washington.

DR. DAVIS: He is also a former member of the Chaba council.

MODERATOR NASH: Next Dr. Gordon Hoople of Syracuse, New York, who is a member of the Committee of Medical Consultants of the Workmen's Compensation Commission of the state of New York, and he is also a member of the Chaba council.

Now last, and I think least, is your moderator. Dr. C. Stewart Nash is a member of the Committee of Medical Consultants of the Workmen's Compensation Committee of the state of New York. He is a consulting otologist to Associated Industries of the state of New York.

May I tell you that all of these men occupy many other positions that are either in or outside the field of occupational hearing loss.

Now question number one to Dr. Hoople.

DR. DAVIS: Before you get going with Dr. Hoople, I would like to throw one at you. What do you mean by "disability" and by "disablement," which you mentioned a while ago?

MODERATOR NASH: It is a little unusual to start by asking the moderator questions, but I think the question is well put. I am going to request you to observe some definitions and to ask questions if you do not understand my meanings.

Disability is partial or complete loss of function or the loss of a member which results in a diminution of earning capacity or of wages. Let's get this point straight. A disability is an impairment in which the worker loses wages. You may recall that the basic compensation laws all through the country have been the same. A worker who lost his leg has not been compensated for the loss of his leg. He is compensated for the loss of wages that resulted from the loss of his leg.

DR. DAVIS: Do I have it right? Is it both the function and the earning power that are gone in the case of disability?

MODERATOR NASH: That is correct. *A disablement is a partial or complete loss of function with no loss of earning power and no loss of wages.* For instance, if a person incurs a loss of hearing by many years' work in a steel plant, truly he can have acquired an occupational hearing loss, but he can work just as well as he ever worked. As a matter of fact, he may work more comfortably with some hearing loss. He has a disablement but he does not have a disability because he has lost no wages.

DR. HOOPLE: These are important definitions, Stew, but they are just definitions, aren't they? Disablement in New York, for instance, is compensable.

MODERATOR NASH: Yes, that is right, but up until 1945 it was not.

Maybe Meyer Fox would like to say something about what happens in Wisconsin with regard to disability and disablement.

DR. FOX: Thus far the various higher courts throughout the country, not only in Wisconsin but also in New York, and the federal courts which cover the Longshoremen's Act, have upheld the right of the worker to collect for a disablement, even though no wage or earning losses have been demonstrated.

MODERATOR NASH: In the state of New York the attorney for the Associated Industries is still working within the courts to counteract the more generous interpretations of the judges. He feels that unless there is loss of earning power, there is no disability. I am afraid he is going to be overruled.

Now let's get to some of the questions.

Dr. Hoople, do you think that an understanding of occupational hearing loss has any significance for the rank and file of otolaryngologists?

DR. HOOPLE: I am sure that it has. We are going to see these cases in increasing numbers. It is true that otolaryngologists in large industrial centers will see more of these patients than those who are in country communities, but I don't believe that any of us are going to be free from seeing some of them. We must be alert to this problem and keep informed, because new things are coming up all the time. We can make ourselves better witnesses when we are asked to testify in court: the modern lawyer, whether he is industry or labor bent, can ask questions that will make us look very foolish if we are not up to date in all the nomenclature and all the definitions.

The rank and file of otologists have a responsibility to the public to diagnose properly the cases that come to them, to be sure that malingerers and those who are trying to feign a hearing loss are not given awards because of erroneous judgments on their part.

I feel that the diagnosis of a hearing loss which is due to exposure to noise is a difficult one to make and it takes all the diagnostic acumen that a man has, but the question that lawyers ask is the one that the physician should be ready to answer: Do you believe with reason-

able medical certainty that this is so? This is the question that you have to face and that you have to be ready to answer.

DR. GLORIG: Gordon, you said "with reasonable medical certainty." Out in California they ask you whether it is possible. What do you answer when they ask you that sort of question?

DR. FOX: Apropos of Aram's question, I am from Wisconsin. We are not expected to be quite certain. All they ask is that we give our opinion based upon reasonable medical probability. In other words, they are not asking us to base our medical opinions and conclusions on an absolute certainty. They are asking us to base them on that evidence in our findings which is so reasonable that it induces belief on our part. That is as far as we have to go.

DR. HOOPLE: The only member of this panel who has not spoken of this question is from Missouri. I would say in answer to this question that you have to be prepared to answer any question that is put to you. That is why I say it is important to be well acquainted with everything that is going on in this field.

MODERATOR NASH: I don't want to prolong this discussion too much. Just remember that if somebody comes into your office as a private patient and you examine him, then he says, "I think I got this loss of hearing out at so-and-so plant," from that point on you are in the problem. You may say, "I am not going to have anything to do with this," but you can be subpoenaed by the compensation court and you can be subpoenaed by the supreme court. So you had better be prepared.

Now shall we go on to the next question? The next question is to Meyer Fox: A man comes into your office complaining of a loss of hearing which he attributes to his work. All your audition tests indicate that he has a high range or perception hearing loss that could be occupational in origin, but it might be a presbycusis, a loss due to mumps or streptomycin or something else.

"Now, Dr. Fox, what do you think? Is it due to my occupation?"

Meyer does not know anything about it, but he speaks to the manager of the plant, whom he knows, and he says, "Do you want me to come out there and look at your plant, and see whether it is possible that you have a noise problem?"

The manager says, "Sure, I'll be glad to have you come out here, but you cannot take any noise level readings."

What are you going to do about it, Meyer?

DR. FOX: First of all, let's be practical. We know that certain types of industrial operations are associated with intense and high noise levels, and that it is quite reasonable to expect hearing loss in the workers in these areas over a sufficiently long period of time. Offhand, we know that these workers include drop forgers, hammermen and helpers, riveters, chippers, pneumatic tool and punch press operators.

In this connection I might refer the audience to a recent publication put out by the Academy, the 1954 Wisconsin State Hearing Survey in which these occupations are classified according to their noise category. This is an excellent piece of investigative work which was done by the Research Center of the Subcommittee on Noise in Industry. Many aspects of the relation of hearing loss to the general as well as the industrial population were studied.

Copies of this publication are available free to Academy members by writing to Dr. Benedict, Secretary of the Academy.

Also published by the Subcommittee is the "Guide for Conservation of Hearing in Noise," which came out in the spring of this year. This guide represents several years' work on the part of the Subcommittee, and in it I think you will find the answer to your question. It has the indications for hearing conservation programs. In other words, an occupation which might produce hearing loss is indicated when the following conditions are present:

First of all, if the worker has had difficulty in communicating by speech while he is in noise, particularly if the workers cannot hear one another with a loud, shouted voice at the distance of a foot.

Second, if these workers keep complaining about tinnitus, ringing in their ears, particularly after working hours.

Third, if they manifest what we call the temporary threshold shift. In other words, they have difficulty in hearing for a few hours after leaving their work but their hearing returns a few hours later.

I think these are practical observations which would indicate to you whether or not they have a noise problem and whether or not there is hearing loss that resulted from the noise exposure.

Of course, for more definite and accurate measures you have to take sound pressure level readings and octave band analyses, and I strongly urge that these readings be made when compensation is in question.

MODERATOR NASH: We will pass to the next question. I want you to follow this question very carefully because an understanding of it will determine whether the patient, the worker, whatever you wish to call him, will or will not get some money fairly soon.

I am going to ask Aram Glorig to talk to you about three terms. He may change or he may alter the order of these terms, but here they are: (1) noise-induced loss of hearing; (2) acoustic trauma; (3) occupational deafness. I want you to listen carefully because he is going to clarify these terms for you. Before you write your report you are going to know what you are talking about; otherwise you are going to be very unfair to your patient.

All right, Aram.

DR. GLORIG: If I didn't know better, I would think I was the Oracle of Delphi the way Stewart talks here. But these terms are extremely important from that standpoint of understanding the problem of noise and its relation to hearing loss. In the first place, the classification which we would like to discuss today would be one that would be headed, "Losses that occur as a result of one's occupation," or the generic term "occupational hearing loss."

There are many types of occupational hearing loss, but there is a way to differentiate them. The first type that Dr. Nash mentioned is noise-induced hearing loss (NIHL). The differentiation of noise-induced hearing loss from the second term, acoustic trauma, is one of *time*. The noise-induced hearing loss is a loss which occurs as a result of long-time exposure to excessive noise. Remember that we are not only talking about the noise itself; we must add a second dimension, which is time. This is extremely important because the long-term, insidious high-tone hearing loss, which we call noise-induced hearing loss, is not due to short exposures of a few hours or a few days or even of a few weeks. It is a long term exposure. Remember this when we talk about noise-induced hearing loss.

Acoustic trauma, on the other hand is different, because an injury to the ear which is known as acoustic trauma is the type of trauma which results from a

sudden impact or impulse-type noise. It is instantaneous in such an impact as might occur from an explosion or from a blow to the head. Here another form of differentiation enters in; the blow to the head causes the same kind of damage to the ear, but it is not due to an acoustic force. It is due to a mechanical force. We are beginning to call such injuries auditory trauma, since they refer to the organ of hearing rather than to the source of the force, which in the case of acoustic trauma is sound, such as an explosive or impact-type noise.

I could mention other auditory trauma, such as the kind that occurs when an ear is exposed to molten slag when a piece of hot metal drops into the middle ear. This could cause a sudden hearing loss, but it is not considered to be acoustic trauma because sound is not the source of its force. It is rather a mechanical, chemical or thermal type of damage.

Is that clear, Stew?

MODERATOR NASH: No, would you give it to us again? I want to know which is the generic term.

DR. GLORIG: In this afternoon's concept it would be occupational hearing loss.

MODERATOR NASH: Okay. Occupational hearing loss, that is the big term. Now we have two subheads, *A* and *B*. What is *A*?

DR. GLORIG: Noise-induced hearing loss, which is the type that occurs as a result of long-time exposure to excessive noise.

MODERATOR NASH: And *B*?

DR. GLORIG: *B* is acoustic trauma which is the result of a short-time, almost instantaneous, type of noise exposure.

MODERATOR NASH: The important thing is that the short term exposure may last a second. A case involving this type of exposure is settled in compensation courts as an accident, whereas in the case of noise-induced hearing loss, you have to establish permanency, which may take a considerable length of time.

Do other members on the board want to contribute anything to this discussion? Let's follow this up a little. Is it true that auditory trauma may assume any type of hearing loss, whereas uncomplicated noise-induced hearing loss is always perceptive? Am I using the term right?

DR. GLORIG: I think so.

MODERATOR NASH: No, I substituted do you understand it?

DR. HOOPLE: Yes, but you omitted acoustic trauma.

MODERATOR NASH: Gordon Hoople, auditory trauma for acoustic trauma.

DR. HOOPLE: All right, the hearing loss from auditory trauma can range all the way from total perceptive loss (a total loss of hearing) to a severe conductive loss. There can be some perceptive loss and some conductive loss. Let's say, for instance, that a man gets a blow on the head. It does nothing to his inner ear but it causes a hemorrhage into his tympanum. He will have a marked conductive loss. That is auditory trauma. If he gets a hot spark in his ear and it burns his drum and does damage to his tympanum, he will have a conductive loss and probably no perceptive loss; but a blow to the head could give him both. He could have the inner ear damages and middle ear changes as well. The noise-induced hearing loss, however, is always perceptive. There is no conductive element in it. This does not mean that noise-induced hearing loss cannot occur in an individual who already has a conductive loss, but the element of the loss that is due to exposure to noise is always perceptive.

MODERATOR NASH: In other words, the short-term assault can result in any or in all types of deafness, but the long-term exposure can only cause a nerve or perceptive type of deafness. Is that correct?

DR. HOOPLE: That is correct. You didn't want me to put in acoustic trauma, but I can perceive where acoustic trauma may be totally perceptive or it may be partially perceptive and partially conductive. A blast may rupture a drum, and this may result in a hearing loss. So that both acoustic and auditory trauma can have a perceptive and a conductive element.

MODERATOR NASH: This new classification is going to be pretty tough on me because I have the terms acoustic trauma and occupational hearing loss written all over the proceedings of the compensation boards and the supreme court of the state of New York. If I get into trouble I shall sue Aram.

Now I am going to talk to Hal Davis. Hal, for years you and I have been fighting over methods of evaluating hearing loss, and when I say fighting I make no understatement. Would you mention some of the methods that we have discussed and over which we have clashed, discussing some of the pros and cons? I would like to have you pay particular attention to the so-called AMA method which I was instrumental in relegating to the bone yard? Okay, Hal.

DR. DAVIS: Stew, I am not going to try to mention each and every method for evaluating hearing loss that has been suggested at one time or another. I am going to speak specifically of only four methods. One of them is very familiar: the so-called Fletcher "point 8" method. That is the oldest. Second is the method recommended by the AMA to which you referred. Then, too, there has developed more recently the New York method. The fourth is the method which has been made official in Wisconsin.

I want to point out that more important than the differences between these methods is a general similarity among all of them that brings out a general principle which seems to be fully accepted. That principle is that the most important consideration in hearing is the overriding consideration of hearing for speech. In one way or another each of these methods undertakes to derive a number to represent hearing loss for speech.

One possible method of getting at this, which has not been used in the medicolegal context although frequently discussed, is speech audiometry, such as the direct use of spondee words. Instead, in practice and historically, we have proceeded by getting a pure tone audiogram, and then by one rule or another calculating from some of the pure tone readings a single number representing the hearing loss for speech. One of the differences among the four (and many other) methods is the choice of frequencies for which the decibel losses are averaged or combined in some way. Perhaps it is the three familiar "speech frequencies," 1000 and 2000 cps. In some cases 4000 is included as well, and in the New York rule, 250 also. In the AMA method four frequencies were used but with different weights: that is, different importance was ascribed to the different frequencies.

I think that one reason the AMA method was discarded, a repudiation Stew accomplished, was the computation of this weighting, as well as the question as to whether the weighting was correct. The computation certainly made the method rather complicated.

Now that is the first step that is common to all methods.

The second step is to get a rule by which to go from the estimate of deci-

bels of loss for speech to some number corresponding to the degree of handicap. That translation is made very simply in the Fletcher and the New York state methods but is made by a much more complicated set of tables and numbers in the AMA method and by rule of intermediate complexity in the Wisconsin method. The details of the rules differ among the different methods, but the important thing that I want to emphasize is the over-all similarity, the general logic of the procedure, first estimating the loss for speech and then, second, a quite independent step, a rule for geting from decibels of hearing loss over to the concept of how much handicap it represents.

DR. HOOPLE: Hal, I think this leaves the audience a little confused because in Florida or in the state of Washington otologists say, "What method do you use in reporting any loss?" This matter is confusing, but research is under way and I hope that somebody will come up eventually with a method that will be universally accepted and universally adopted. I recall saying in this auditorium three years ago that this research was under way and maybe we would have the end result in two years. The answer is not ready yet. I am not going to be foolhardy enough to say when, but at least I think everybody should be aware that serious work is going on toward the adoption of a plan that will be universally accepted.

MODERATOR NASH: I am going to direct a question to Dr. Hoople and to Dr. Fox. I don't want them to go into too much detail.

Gordon, I would like to have you tell me about this six-month waiting period, what it means and how it works in New York state. Then I am going to ask Meyer the same question about the plan in Wisconsin.

DR. HOOPLE: In New York the committee of medical consultants to the compensation commission felt that they should put in their regulations that there should be a six-month waiting period. This waiting period means that before the worker's hearing can be properly assessed there must be a six-week period from the date of his last exposure to noise.

MODERATOR NASH: Six months, not six weeks. The six-month postulate is my project. I don't want you to misname it.

DR. HOOPLE: Six months, excuse me. it shows how when you are thinking of some main principle you can skip the details. I am sorry. It is six months. In other words, the worker must be out of his noisy environment for six months before judgment can be made as to the permanency of his hearing loss. We know that in addition to his permanent loss, the worker may have a temporary and reversible loss. This is known as Temporary Threshold Shift. For this reason, a recommendation was made to the compensation commission in New York that a waiting period of six months be put in the regulations, and such a ruling now exists in New York.

MODERATOR NASH: I think you will find that the six-month waiting period, the so-called "six-month postulate," is served without dressing in the states of New York and Wisconsin. Meyer, will you tell us about Wisconsin?

DR. FOX: I don't know what you mean by being served with and without dressing. Actually what happened in Wisconsin was that the Medical Advisory Committee to the industrial commission was in no position to say that the recovery period should be six months, three months or three days. However, the Medical Advisory Com-

mittee was willing to go along with the six-month postulate and say that hearing loss which persisted after six months removal of the individual from the noise could be considered as permanent. However, in Wisconsin the advisory committee to the industrial commission—that body which includes representatives from labor and industry—showed great wisdom in putting into the law (and this by legislative action) that a worker could file a claim six months after any of the following events provided he remains away from injurious noise for that length of time: (1) termination of employment, (2) retirement, (3) transfer from noise into a quiet area, (4) layoff or a prolonged strike for one year. The thing I want to point out is that this is a social and economic decision and not a medical decision, and now from what I have heard in New York and elsewhere they would like to follow this type of reasoning.

MODERATOR NASH: I have the greatest admiration for what has been accomplished in Wisconsin. Meyer Fox has done a perfectly splendid job. I am just kidding him.

Now we have a few more questions, a couple of quickies. I am going to ask Aram two and I am going to ask Hal Davis two.

Aram, what noise level is necessary to produce a hearing loss?

DR. GLORIG: I am not sure about the answer. I think that before we talk about what levels or what noise or how much of which, we should explain here that we are not talking about noise only. We are talking about noise exposure, and we should always ask how much noise exposure is necessary rather than what noise level or how much noise.

We ought to divide this noise exposure into three main headings: (1) how much noise; (2) what frequency; and (3) amount of exposure (how many hours a day or how many years during a lifetime). When we can answer all of these questions, we can come up with a fairly decent reply, but until then we have chosen to express this in our guide as follows:

> At the present time our knowledge of the relations of noisy exposure to hearing loss is much too limited for us to propose safe amounts of noise exposure. We can, however, point to certain noise levels that indicate when it is advisable to initiate hearing conservation programs. These levels will not be general because a different level is needed for different types of noise and different schedules of exposure. The hearing conservation level that we now specify tentatively applies only to years of exposure to broad bands, steady noises, with relatively flat spectrum. It does not apply to short exposures and, above all, it does not apply to impact noises or explosive type noise.

This tentative hearing conservation level is stated as follows:

> If the sound energy of the noise is distributed more or less evenly throughout the eight octave bands and if a person is to be exposed to this noise regularly for many hours—I want you to get that—many hours a day five days a week for many years then, and only then, if the noise level in either the 300 to 600 bands or the 600 to 1200 bands exceeds 85 db the initiation of noise exposure control and tests of hearing is advisable. The more the active band level exceeds 85 db, the more urgent is the need for hearing conservation.

One last important statement:

> The 85 db is not an over-all sound pressure level. It refers only to two particular octave bands. The over-all level of noise is always higher than the level in any one octave band. In typical industrial steady noises it may be as much as 20 db higher.

Is that clear, Stew?

MODERATOR NASH: If it isn't we can read it in the Guide when we get home. Your presentation was excellent.

One more, Aram. What frequencies do you think are necessary for an in-

dustrial audiometer, that is, the way they do it in the factory? What frequencies should they test?

DR. GLORIG: In other words, what frequencies should we use if we are running a hearing conservation program in which the audiometer is to monitor our hearing conservation program? We have found by experience that it is only necessary to measure 500, 1000, 2000, 3000, 4000 and 6000. We are not excluding the other frequencies. We say that these are the only frequencies that are necessary for monitoring purposes as differentiated from many frequencies or as many as you wish for diagnostic purposes.

MODERATOR NASH: Now we have two quickies that we are going to ask of Dr. Davis.

First, define "hearing loss" and, second, define the "normal hearing level."

DR. DAVIS: We all talk about hearing loss but I don't think that many of us realize how ambiguous and confusing this term can be; in other words, how many different meanings have become attached to it. I believe that this is an important point because it is precisely in this context of occupational hearing loss and disablement and disability that the confusions from the multiple meanings of the term really catch us by the heels.

I would like to suggest that we restrict the term hearing loss to just one of the three different meanings that I shall give you.

First, we have the *symptom:* the thing that the patient suffers from and complains of. I will put that down as the meaning for which the term will be retained, as when we speak of a "conductive hearing loss" or a "perceptive hearing loss." This is good, well established usage. Second, we have the thing that is measured on the audiometer, the number that expresses the *status* of the subject's hearing. The dial on the audiometer is labeled "hearing loss," but this, as I think you will see in a moment, leads to confusion. I suggest that when we mean the thing which is measured on the audiometer and that expresses the status of the hearing, we should speak of *hearing level.* More fully stated, the status of hearing is measured by finding the sound level that is just at the threshold of hearing.

Now the third meaning, of course, is a change: a change in status. A person's hearing gets worse. We say that he suffers a hearing loss. We can translate this quite easily and simply into the words, "He suffers a loss of hearing." The rewording emphasizes the fact that we are talking about a change. This is logical enough by itself, but now if you say that a patient "has a hearing loss of 25 decibels," that implies to the layman that the patient once had something that he has now lost. That implication keeps creeping in unavoidably. Now this may not be true. This patient may never have had a hearing level better than 25 db. So I will introduce another term to specify a change of hearing. It is the term used by Dr. Glorig and his group. It is *threshold shift.* This clearly means *a change in the status of hearing* one way or another. It emphasizes the change.

Of course, a threshold shift may be brought about by exposure to noise. This is now a noise-induced hearing loss. The hearing loss is a symptom or a condition. The threshold shift may be temporary or it may be persistent, and that is part of the problem.

Getting back to your other question, Stew, now that I have my terms lined up here — What is the normal hearing level? I am interested that you wrote "level" without any of this discussion in advance. What is the normal reference level at which we start our scale of hearing level? This refers, of course, to the

zero on the audiometer scale or, putting it the other way, the audiometer scale is presumably based on a representative hearing level that is characteristic of people who have not suffered from any particular insult to hearing or otologic disease.

Here we must make a statistical or "survey" sort of approach. For our present purposes we can say that the central tendency of this grouping is taken as the zero of the audiometer. There is some question as to whether this is really the best reference level. Perhaps we should use the somewhat better hearing level that we get in the laboratory, instead of taking thresholds of people when they come into the office or when they are tested in Glorig's survey at the Wisconsin State Fair. The latter gives one collection of hearing levels. In the laboratory we get a somewhat better and more sensitive collection. Here we use a bit of training and coaching of the subjects, and they have good motivation. These two sets of "normal thresholds" are not quite the same and I think we should regard this as an open question as to what our long term view on this should be.

But the one additional point I would like to make is that "normal" in this context should be thought of as a *range*. We get one number that is the central tendency. That should be given first; but we should also think of a spread on both sides. This is at least 10 db plus or minus. Somewhere about plus 15 db we pass from the normal range to the hearing levels that are really significantly different.

MODERATOR NASH: I think that is pretty good.

May I call to your attention again—I have already called your attention twice so far—to the importance of having in your office the Guide for Conservation of Hearing in Noise. This manual is put out by the Subcommittee on Noise in Industry, and you may get it by addressing Aram Glorig at the Research Center, the Subcommittee on Noise in Industry, 111 North Bonnie Brae Street, Los Angeles 26, California. I advise you to have it in your office at all times.

May I also call your attention to the 1954 Wisconsin State Fair Hearing Survey, a statistical treatment of clinical and audiometric data. That manual is also obtainable through Aram. You should have it in your library.

TRANSACTIONS

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INTERNATIONAL ASSOCIATION OF SECRETARIES OF OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETIES

History of Medicine and the Association of Secretaries of Ophthalmological and Otolaryngological Societies

HISTORY of medicine and science is part of the history of civilization through the ages. It can provide an additional bridge of understanding between ophthalmologists and otolaryngologists throughout the world.

The sum total of any advanced culture is an enormous conglomerate of experiences; of knowledge, of beliefs, of institutions, of practices, the elements of which belong to the most different strata of the past. In order that we may call our present-day civilization modern, newer trends become emphasized and equipped with additional features more acceptable to the changing taste.

The most momentous event with regard to the progress of science certainly was the invention of glass lenses. Prepared theoretically in the eleventh century by Ibn el Haitham, reading spectacles were actually produced at the close of the thirteenth century in upper Italy and their use became fairly widespread, especially during the fifteenth and sixteenth centuries. This must have become an important factor in increasing the number and the ages of people participating in learning. The appearance of glass lenses had a tremendous impact, however, when around 1600, it made possible the invention of the telescope (Galileo, Kepler) and in the second half of the seventeenth century, the invention of the (composite) microscope (Leeuwenhoek). Thus the never-ending journey was started in two opposite directions, into the discoveries of the world of the infinitesimally small and of the infinitely far, steadily widening the horizon of mankind.

Let us try to answer the question: what does history of medicine and science mean to us? Every educated person is supposed to know the past of his own country in addition to the fundamentals of general history. Otherwise he would be unable to comprehend intelligently its traditions, its contemporary trends and institutions—all requirements for good citizenship. The same necessarily applies to the need of a working knowledge of

how one's special branch of work has developed. History of medicine and its specialties has nowadays become one of the many branches of the great tree of the history of sciences, arts and techniques.

Knowledge of the past teaches us two cardinal lessons: (1) not to commit the common error of overestimating the almost unheard-of progress, especially in the technical field, made in our own times, and (2) not to underestimate earlier times. In other words, history teaches us to become critical and tolerant. In the past, even in antiquity, scientifically correct views on medicine and science have been brilliantly expressed by an elite of men who only were unable to prove them (think — in astronomy — of the famous heliocentric hypothesis by Aristarchus of Samos in the third century B.C. combined with the assumption of the rotation of the earth about its own axis!); correct questions have been asked which could not be answered for lack of adequate technical means.

Some ophthalmologic examples, taken from the so-called Aristotelian Problems (not genuine, but very early): "Why do the myopic write small letters?" "Why do the myopic squeeze their eyelids together in order to see (well)?" "Why is it, that in the two kinds of weakness of the eyes, the myopic and the senile, does the one, when he likes to see (well), approach (to), and the other one, hold far away (from the eye)?"

At all periods of history there were men who, much ahead of their time, pronounced sound original ideas that did not impress their contemporaries or were rejected by them. Their intuitive progressive conceptions or inventions had either to be rediscovered centuries later or to remain in the shadow for long periods until recognized by later generations.

The ancients thought the lens to be the central organ of vision, situated in the center of the eyeball. *In praxi* it therefore was unthinkable for the coucher (for cataract) that it was the lens that had to be attacked (and frequently even injured) in an operation aimed at the restoration of vision. So hypochyma, as cataract was called by the Alexandrians, having nothing to do with the lens, had to be some inspissated matter developing behind the pupil. Even after Fabricius ab Aquapendente had, in 1600, demonstrated the correct position of the lens (subsequently to dissection of a frozen eyeball), and Kepler, in 1604, discovered its true function, cataract continued to be some solidified matter in the "empty space" between the pupil and the crystalline lens. It took another century until Brisseau (in 1705) definitely proved, by autopsy, that the cataract dislocated by depression was the dislocated opaque lens.

"Hardness of the eyeball," the main characteristic of glaucoma, discovered in 1585 by Jacques Guillemeau (whose book was translated into English by Banister in 1622) had to be rediscovered by J. Z. Platner (*"oculus durus, digito renitens"*) in 1745.

After the revolutionary innovation (in 1748) by a daring man of genius—Jacques Daviel—of *extracting* the cataract from the eye instead of the ancient, risky and ill-reputed method of couching, it is surprising to find that it took another century until couching was definitely given up as a rival method of extraction, and this in spite of Daviel's successes, then quite unusual, in some 89 per cent of his cases. The reason was mainly conservatism and obstruction on the part of a large section of the profession while, it is true, a good deal of dexterity was required from the surgeon in handling the eye and the patient; and there was the undeniable danger of

infection. It is noteworthy, however, that the lengthy discussions of the pros and cons had at least one positive result: procedures were elaborated how best to deal with soft cataracts by discission.

"Alles Gescheit ist schon gedacht worden—man muss nur versuchen, es noch einmal zu denken" [Everything clever has already been thought, one has only to try to think it once again], said Goethe. Therefore, no new discovery is altogether new in the strict sense of the word, and hence "the precursors of ideas." Think of the astonishingly realistic ("modern") Hippocratic approach to medicine in the fifth century B.C.!

The conception of the "healing power of nature," pronounced already by Hippocrates, had to be rediscovered in the late Middle Ages. The wise dictum of Ambroise Pare, the eminent surgeon of the sixteenth century: "I dressed him and God cured him," was not merely a pious phrase — it was a deep biologic conviction. This fundamental idea had slowly to fight its way through until Sydenham (the "modern Hippocrates" of the seventeenth century) emphasized more clearly: "A disease, in my opinion, however prejudicial its causes may be to the body, is no more than a vigorous effort of nature to do away with the morbific matter, and thus recover the patient."

Thus, there exists a close interrelation between contemporary medicine and its past. Every experienced older practitioner has seen in his lifetime peculiar trends in medicine, which one may safely call fashions. These were at first received enthusiastically, but after some time elapsed were found to be of no use at all, and have sometimes been discarded after having done, in practice, more or less harm. In our present period of sensational developments in medicine, these undulating and transient fashions —just because of the speed with which they come and go—have been witnessed even by younger members of the profession. Everyone now knows the limitations of the sulfa drugs, the shady side of the otherwise powerful treatment with antibiotics, the changing views on correct nutrition.

History-mindedness has a bearing not only on the critical sense of the medical man but also on his ethical behavior. Knowledge of the limitations of human judgment through emotional narrowness, ignorance and prejudice of man may prove its value.

Exploration of new avenues in science makes sense only when brought together with what we possess and know to have inherited from our ancestors. Let us use this information for increased understanding of our own work and of that of our colleagues here and abroad.

Professor Aryeh Feigenbaum
President,
Israel Ophthalmological Society

In Memoriam

Richard Parker Bell, Jr. ..... Lakewood, Ohio
July 26, 1958

Leopold Benno Bernheimer ..... Chicago, Illinois
July 15, 1958

Irving Myron Birenboim ..... Kansas City, Missouri
July 24, 1958

Guy Lee Boyden ..... Portland, Oregon
September 9, 1958

Charles Howard Gordon ..... Portland, Maine
July 28, 1958

John McChesney Hogshead ..... Chattanooga, Tennessee
July 13, 1958

Jaroslav H. Hulka ..... Long Island, New York
June 18, 1958

Cecil Frederick Johnston ..... Bluefield, West Virginia
July 21, 1958

Lowell Waldo King ..... Salem, Ohio
June 29, 1958

Henry Kirkpatrick ..... Yateley, Hampshire, England
May 11, 1958

Fritz Lassen ..... Pueblo, Colorado
June 30, 1958

Frank Wilmore Poindexter ..... Newport News, Virginia
July 4, 1958

Francis S. Schwarz, Jr. ..... Shaker Heights, Ohio
August 31, 1958

Russell Hope Strange ..... Mount Pleasant, Michigan
June 26, 1958

Edwin Forbes Tait ..... Norristown, Pennsylvania
September 23, 1958

Ricardo Tapia y Fernandez ..... Mexico, D. F.
September 3, 1958

Necrology

IN MEMORIAM

October 1957 - October 1958

Eldridge Stevens Adams, Inglewood, Calif.
Arthur Francis Ash, Weehawken, N. J.

Otto Barkan, San Francisco, Calif.
August Leo Beck, New Rochelle, N. Y.
Richard Parker Bell, Jr., Lakewood, Ohio
Leopold Benno Bernheimer, Chicago, Ill.
Irvin Myron Birenboim, Kansas City, Mo.
William D. Black, St. Louis, Mo.
Guy Lee Boyden, Portland, Ore.
Carl Frederick Breisacher, Charleston, W. Va.
Moses Lewis Breitstein, Baltimore, Md.
Daniel Webster Brickley, Marion, Ohio
Harry Francis Byrnes, Springfield, Mass.

Linus Samuel Cave, Syracuse, N. Y.
Carl Clarence Chase, Middletown, Conn.
Leon Clinton Combacker, Fergus Falls, Minn.
Clyde Robert Cowan, Buzzards Bay, Mass.

Elmo Eugene Dillon, Homewood, Ill.
Edward Philip Drescher, Berkeley, Calif.
George Silas Duntley, Macomb, Ill.
Howard Victor Dutrow, Dayton, Ohio

Howard Daniel Fallows, Mason City, Iowa
Ivan Fawcett, Wheeling, W. Va.
Alexander Feher, New York, N. Y.
Everett Russell Ferguson, Cleveland, Tenn.
Maxwell Fineberg, St. Louis, Mo.

Jerry M. Garber, Mansfield, Ohio
Charles Howard Gordon, Portland, Maine
Thomas Francis Gowen, Costa Mesa, Calif.
Edwin Albert Griffin, Brooklyn, N. Y.
Milus Liddell Gunn, Harlan, Ky.

Harry H. Haggart, Cincinnati, Ohio
Alfred William Haskell, Portland, Maine
William Jacob Hertz, Allentown, Pa.
John McChesney Hogshead, Chattanooga, Tenn.
Jaroslav H. Hulka, Long Island, N. Y.
Vesse Reeves Hurst, Longview, Texas

Chevalier Jackson, Schwenkville, Pa.
Hobart Clemens Johnson, Mankato, Minn.
Cecil Frederick Johnston, Bluefield, W. Va.
Alexander Spencer Kaufman, Philadelphia, Pa.

Frank Conrad Keil, New York, N. Y.
Peter Augustus Keil, Brooklyn, N. Y.
Lowell Waldo King, Salem, Ohio
Henry Kirkpatrick, Yateley, Hampshire, England
Alan DeGrief Knisely, Lima, Ohio
Heinrich Franz Guenther Kobrak, Clarkston, Mich.
Hugh Alva Ross Kuhn, Hammond, Ind.

Fritz Lassen, Pueblo, Colo.
Richard Ormond Leavenworth, St. Paul, Minn.
Albert Napoleon Lemoine, Sr., Kansas City, Mo.

Wayland Hoyt Maloy, Shenandoah, Iowa
John Hollis Matheson, Des Moines, Iowa
Willis Leslie McConkie, Carroll, Iowa
Joseph Letelle McCool, Monrovia, Calif.
James Harold Mendel, Coral Gables, Fla.
Frederick William Merrifield, Wilmette, Ill.
H. Edward Miller, St. Louis, Mo.
Harold Rexford Miller, New York, N. Y.
H. F. Minor, Memphis, Tenn.
William Henry Minton, St. Joseph, Mo.
William Mithoefer, Cincinnati, Ohio
William F. Molt, Indianapolis, Ind.
Daniel Cameron Montgomery, Greenville, Miss.
David Jacob Morgenstern, Brooklyn, N. Y.

Edmonde De Witt Neer, New York, N. Y.
Fritz Nelson, Colorado Springs, Colo.
Max Nisselson, New York, N. Y.

Carl M. Oberg, Minneapolis, Minn.
Dennis Francis O'Connor, South Orange, N. J.

Henry Boylan Orton, Newark, N. J.
Louis Ostrom, Rock Island, Ill.

Theodore Felix Paprocki, Ogdensburg, N. Y.
Sir John Herbert Parsons, London, England
Clarence N. Peeler, Charlotte, N. C.
John Chaney Peterson, Lincoln, Neb.
Frank Wilmore Poindexter, Newport News, Va.
Ralph Alex Poirier, Birmingham, Mich.
Lawrence Tyler Post, St. Louis, Mo.

Robert Linton Ralston, Spartanburg, S. C.
Morris Munice Rossman, Freeport, N. Y.
Martin Van Buren Russell, ElDorado, Ark.
Linus Martin Ryan, St. Louis, Mo.

Francis S. Schwarz, Jr., Shaker Heights, Ohio
Isadore Marwell Shapero, Rochester, N. Y.
Relza Newton Sherman, Bradenton Beach, Fla.
Olga Lazarovna Sitchevska, New York, N. Y.
William Steinman, Miami, Fla.

Howard Leigh Stitt, Cincinnati, Ohio
Arthur Leslie Stotter, Cleveland, Ohio
Russell Hope Strange, Mount Pleasant, Mich.

Edwin Forbes Tait, Norristown, Pa.
Ricardo Tapia y Fernandez, Mexico, D.F.
Martin Clever Taylor, Pittsburgh, Pa.
Charles Alston Thigpen, Montgomery, Ala.
Francis W. Thomas, Columbus, Ohio
Henry Eagle Thomason, Summers, Ark.
Baxter Hall Timberlake, Atlantic City, N. J.
Everett Hale Tomb, Framingham Centre, Mass.
Gabriel Frederick Tucker, Rosemont, Pa.

Oscar Lloyd Veach, Sheridan, Wyo.
Charles Bernard Voigt, Mattoon, Ill.

Hugh Claibourne Wolfe, Greensboro, N. C.
Doris Lee Surles Woolsey, St. Louis, Mo.
Joseph William Wright, Indianapolis, Ind.

Charles A. Young, Sr., Roanoke, Va.

William Zentmayer, Merion Station, Pa.

ELDRIDGE STEVENS ADAMS

Elridge Stevens Adams was born in Atchison, Kansas, on November 30, 1891. He attended the University of Kansas and received his medical degree from Rush Medical College in 1914. He began his medical practice in Trinidad, Colorado, in 1916. Dr. Adams was a charter member and a vice president of the Aero Medical Association, and was a reserve officer in the military service from 1917 until his retirement as a colonel in the Air Force in 1951.

Dr. Adams was graduated from the School of Aviation Medicine, Brooks Field, Texas, and was named assistant chief of the medical section of the Bureau of Air Commerce, retaining this position until 1933 when he was named aviation medical advisor to the Republic of China. In 1937, he returned to the Department of Commerce as chief medical officer of the Bureau of Air Commerce. He was ordered to active duty in 1940 as a flight surgeon and served with the Army Air Force until 1946, at which time he became regional medical officer for the CAA in Los Angeles. For the past four years he was western regional physician for American Airlines.

Dr. Adams was a specialist certified by the American Board of Otolaryngology, a member of the founders' group in aviation medicine of the American Board of Preventive Medicine and was elected a fellow of the Aero Medical Association when this grade of membership was created in 1942. At one time, Dr. Adams held a private pilot's license. He was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1920 and on January 1, 1950, he became a life member of this organization.

Dr. Adams died of coronary occlusion on October 18, 1957.

ARTHUR FRANCIS ASH

Arthur Francis Ash was born in Gananoque, Ontario, Canada. He was a diplomate of the American Board of Otolaryngology. He received his Doctor of Medicine degree from Queens University Faculty of Medicine, Kingston, Ontario, in 1901. He began the practice of medicine in Duke Center, Pennsylvania, later

moved to Bedford, Pennsylvania, and eventually became assistant house surgeon at Manhattan Eye, Ear, Nose and Throat Hospital. He became associated Hague Maternity Hospital, Jersey City, New Jersey, in 1925 and remained on the staff of this hospital until his retirement in 1957. He also worked at the Margaret Hague Maternity Hospital, Jersey City, and St. Mary's Hospital, Hoboken. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons and served as a medical service examiner in both World Wars.

Dr. Ash was one of the oldest physicians in Hudson County when he died March 9. He is survived by his wife, a son and a daughter.

OTTO BARKAN

Dr. Otto Barkan, a diplomate of the American Board of Ophthalmology, was graduated from the University of Munich School of Medicine in 1914. He was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the Association for Research in Ophthalmology. He served on the faculty of Stanford University School of Medicine and, in 1954, the Section on Ophthalmology of the American Medical Association awarded him the Lucien Howe Medal for research in ophthalmology and allied branches. Dr. Barkan was a veteran of World War I, and was associated with St. Joseph's and Stanford University hospitals. He died on April 26, of cerebral hemorrhage.

AUGUST LEO BECK

Dr. August Leo Beck of New Rochelle, New York, attended New York University and Bellevue Hospital Medical College, receiving his medical degree in 1908; he took further training at New York Post-Graduate Medical School and worked with Drs. Chevalier Jackson and Mosher in endoscopic studies at Harvard Medical School. From 1909 to 1922 he was an instructor and assistant professor of ophthalmology at New York Post-Graduate Medical School.

Dr. Beck belonged to the American Medical Association and the New York Academy of Medicine. He was a fellow of the American College of Surgeons, American Laryngological Society, American Laryngological, Rhinological and Otological Society. He served during World War I in the medical corps of the U. S. Army. He practiced for 45 years in the New Rochelle Hospital; he was the director of otolaryngology and ophthalmology for the past 25 years.

He was born in New York, June 25, 1883, and died in New Rochelle, July 6. Surviving him are his widow, Ella W. Beck, and a brother.

RICHARD PARKER BELL, JR.

Richard Parker Bell, Jr., received his Doctor of Medicine degree from Western Reserve University in 1945; he was certified by the American Board of Ophthalmology in 1951 and was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1952. He practiced his specialty in Cleveland, Ohio.

Dr. Bell was a member of the Associatino for Research in Ophthalmology, the Pan-American Association of Ophthalmology, the American College of Surgeons and the American Medical Association. He died July 26.

LEOPOLD BENNO BERNHEIMER

Leopold Benno Bernheimer was graduated from the University of Illinois College of Medicine in 1922; he was certificated by the American Board of Otolaryngology in 1930.

Dr. Bernheimer practiced most of his life in Chicago, Illinois. He was on the staff of Michael Reese Hospital and was associated with Cook County Hospital and the Veterans Administration Hospital in Hines.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the American Laryngological, Rhinological and Otological Society and the American College of Allergists.

Dr. Bernheimer died July 15, aged 61, of carcinoma of the rectum and diabetes mellitus.

IRVIN MYRON BIRENBOIM

Irvin Myron Birenboim received his premedical education at Washington University in St. Louis, Missouri. He was graduated from the Washington University Medical School in 1944. Shortly

thereafter, he began practicing in Kansas City, Missouri.

Dr. Birenboim was certificated by the American Board of Otolaryngology in 1951 and soon became a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was also a member of the American Medical Association, the Kansas City Society of Ophthalmology and Otolaryngology and the Kansas City Southwest Clinical Society.

He had been an instructor in otolaryngology at the University of Kansas Medical Center, a staff member of Menorah Hospital Medical Center and St. Mary's Hospital and attending otolaryngologist at the Kansas City Municipal Hospital and Alfred Benjamin Dispensary.

Dr. Birenboim became an Inactive Fellow of the Academy shortly before his death July 24.

WILLIAM D. BLACK

Dr. William D. Black was graduated from Marion Sims College of Medicine, St. Louis, Missouri, in 1896. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology.

During his medical career, Dr. Black served on the faculty of Barnes Medical College and on the staff of the Missouri Baptist Hospital. His practice began in general medicine and surgery; he then practiced obstetrics; and finally he devoted his full time to diseases of the ear, nose and throat.

Dr. Black was born in St. Louis, October 7, 1874. He died in St. Louis, December 2, 1957. Mrs. Olga Endres Black, his widow, survives.

GUY LEE BOYDEN

Guy Lee Boyden of Portland, Oregon, was graduated from Northwestern University School of Medicine in 1911. He was certificated by the American Board of Otolaryngology in 1926. Most of his professional life was spent in Portland, Oregon.

Dr. Boyden served as clinical professor of otology, rhinology and otolaryngology at the University of Oregon Medical School. He was a member of the Pacific Coast Oto-Ophthalmological Society, the Oregon Academy of Ophthalmology and Otolaryngology, the American Medical Association and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

At this year's meeting, Dr. Boyden was to receive the Academy's Award of Merit for appreciation of services to the educational program of the Academy. He died September 9, aged 73.

CARL FREDERICK BREISACHER

Carl Frederick Breisacher was born July 30, 1911, in Altoona, Pennsylvania. After receiving his degree of Doctor of Medicine from Jefferson Medical College of Philadelphia, Dr. Breisacher interned for two years at Episcopal Hospital in Philadelphia. He remained in the city as a resident in ophthalmology at the Wills Eye Hospital.

He was certified by the American Board of Ophthalmology in 1944 and, in 1946, began practicing in Charleston, West Virginia. Dr. Breisacher was on the staffs of Herbert J. Thomas Memorial Hospital in South Charleston, Charleston General, Memorial, and Kanawha Valley hospitals. He was a fellow of the American College of Surgeons, a member of the American Medical Association, and the West Virginia Academy of Ophthalmology and Otolaryngology.

Dr. Breisacher died October 16, 1957, in Charleston. He is survived by his wife, Mildred, and daughter, Nancy.

MOSES LEWIS BREITSTEIN

Dr. Moses Lewis Breitstein was graduated from The Johns Hopkins University School of Medicine, Baltimore, Maryland, in 1919, and was certified by the American Board of Otolaryngology. He was associate member of the American Medical Association; a fellow of the American College of Surgeons; an otologist for the city health department; a consultant in otology for the Maryland State Department of Education and president of the Baltimore Hearing Society; and he was associated with the Hospital for Women of Maryland, the Baltimore Eye, Ear and Throat Hospital and the Union Memorial Hospital.

Dr. Breitstein died July 20, 1957, of coronary heart disease. He is survived by his widow, Mrs. Dorothy L. Breitstein; a daughter, Dorothy Breitstein; a son, Charles Breitstein, and two grandchildren.

DANIEL WEBSTER BRICKLEY

Daniel Webster Brickley was born January 30, 1877, in Galion, Ohio. He died April 15, in Marion, Ohio, of tuberculosis.

Dr. Brickley attended Ohio Medical College and received his medical degree from that school in 1904. He had additional training in Budapest, Vienna. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, past president of the Marion County Academy of Medicine and served on the staff of the Marion General Hospital.

Dr. Brickley is survived by his widow and two sons, Daniel W. Brickley, Jr., M.D., Marion, Ohio, and Lester Brickley, Dayton, Ohio.

HARRY FRANCIS BYRNES

Dr. Harry Francis Byrnes, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died January 2, in Springfield, Massachusetts, of bronchopneumonia.

Dr. Byrnes was born October 9, 1877, in Worcester, Massachusetts. He received his medical degree from the Baltimore Medical College in 1904, and took further training at the University of Vienna, Vienna, Austria, and at the Royal Ophthalmic Hospital in London. He practiced general medicine and surgery in Westfield, Massachusetts, from 1905 until 1909, when he moved to Springfield, Massachusetts, and limited his practice to diseases of the eye, ear, nose and throat. He was on the staff of Mercy Hospital in Springfield, and while at this hospital served as chief of the eye, ear, nose and throat service and as president of the staff. He was president of the Springfield Academy of Medicine in 1937.

Dr. Byrnes was certified by the American Board of Otolaryngology; was a fellow of the American College of Surgeons; a veteran of World War I, and was associated with Wesson Memorial and Springfield hospitals as well as Mercy Hospital in Springfield.

Dr. Byrnes was a pioneer motorist and an automobile fancier. He was president of the Springfield Automobile Club in 1939 and was on the board of directors at the time of his death. He was noted for his philanthropic work in western Massachusetts; he received the honor of being made a Sovereign Knight of the Order of Motta by Pope Pius XII in 1941.

LINUS SAMUEL CAVE

Linus Samuel Cave, a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, was born December 19, 1888 at Ohio, New York. He attended Syracuse University and received his degree of Doctor of Medicine from the Syracuse University College of Medicine in 1912. He interned at St. Joseph's Hospital in Syracuse and went into practice in that city.

In 1925, Dr. Cave became a resident at Massachusetts General Hospital and later studied in Vienna. He was certified by the American Board of Otolaryngology in 1928. Dr. Cave was clinical professor emeritus of otolaryngology at his alma mater and was on the staffs of the City Hospital, St. Joseph Hospital, Syracuse Memorial Hospital and Syracuse Psychiatric Hospital.

He was a member of the American Medical Association and the Central New York Eye, Ear, Nose and Throat Society. Dr. Cave died August 10, 1957 at Amber, New York. He is survived by his wife, Elly Katherine, his son, Dr. Linus W. Cave, and four grandchildren.

CARL CLARENCE CHASE

Carl Clarence Chase, an EENT consultant at Connecticut State Hospital and the senior attending otolaryngologist and ophthalmologist at Middlesex Memorial Hospital, died in Middletown, Connecticut, September 17, 1957.

He was born November 29, 1900, in Vermont, and received the degree of Doctor of Medicine from the University of Vermont College of Medicine in Burlington in 1924. Dr. Chase was a resident at Newark Eye and Ear Hospital in 1930 and certified by the American Board of Ophthalmology in 1938. He was a member of the Central Medical Society in Middletown and the American Medical Association.

Dr. Chase is survived by his wife, Dorothy, one son, Donald, two daughters, Caroline and Elizabeth Ann, and eight grandchildren.

LEON CLINTON COMBACKER

Leon Clinton Combacker was born January 10, 1883 in Osceola, Wisconsin. He received his medical degree from the University of Michigan Department of Medicine and Surgery in 1909 and did postgraduate work at the University of Minnesota. He was certified by the American Board of Ophthalmology in 1928;

was a member of the American Medical Association, and in 1935 he was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Combacker died October 1, 1957, in Fergus Falls, Minnesota. He is survived by his wife, Mabel A.; two daughters, Catherine Hughes of Wilmington, Delaware, and Alice Olson of Zumbrota, Minnesota; and five grandchildren.

ROBERT CLYDE COWAN

Robert Clyde Cowan, a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, died May 10, aged 74.

Dr. Cowan practiced otolaryngology in Boston for many years. He was graduated from Middlesex College of Medicine and Surgery, Cambridge, Massachusetts, in 1915. He was a member of the American Medical Association.

ELMO EUGENE DILLON

Dr. Elmo Eugene Dillon served Homewood, Illinois, as an ophthalmologist and otolaryngologist for 28 years. He began practicing there after being graduated from Northwestern University Medical School in 1926.

Dr. Dillon taught ophthalmology at Northwestern University Medical School, was a staff member at Ingalls Memorial Hospital, Harvey, Illinois, and St. James Hospital, Chicago Heights, Illinois, and attending ophthalmologist at Cook County Infirmary in Oak Forest, Illinois.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association and the Chicago Ophthalmological Society. He received the Medal of Honor from the government of Haiti for medical and advisory services to that country.

Dr. Dillon died May 13, survived by his wife, Beulah, and their daughter, Ann.

EDWARD PHILIP DRESCHER

Edward Philip Drescher attended the University of California from 1932 to 1940, receiving degrees as a Bachelor of Arts and Doctor of Medicine. He interned at the San Francisco County Hospital from July 1939 to July 1940. During World War II he served in the medical corps of the U. S. Army. He was a fellow in ophthalmology at the Mayo Foundation, Rochester, Minnesota, from April 1947 until April 1950, and since that time he had been practicing his specialty in Berkeley, California, where he was on the faculty of the University of California and was chief of ophthalmology at the Herrick Memorial Hospital.

Dr. Drescher was certified by the American Board of Ophthalmology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association and the Alumni Association of the Mayo Foundation.

Dr. Drescher was born April 11, 1915, in San Francisco. He died in Berkeley, July 12.

GEORGE SILAS DUNTLEY

Dr. George Silas Duntley, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died October 14, 1957, at Peoria, Illinois.

Dr. Duntley was born in Bushnell, Illinois, June 25, 1879. He attended the Northwestern College of Pharmacy, and was awarded a Doctorate in Philosophy from that school in 1902. He was graduated from the Northwestern College of Medicine in 1908. He was a member of the original board of governors of the American Chapter of the International College of Surgeons; a fellow of the American College of Surgeons; a member of the American Medical Association.

Dr. Duntley was interested in photography and flying. He became interested in flying in 1934, at the age of 55, and for many years he piloted his own plane.

Dr. Duntley is survived by his widow, Nola Seibert Duntley; his son, Dr. Quimby Duntley of La Jolla, California, and three grandchildren.

HOWARD VICTOR DUTROW

Howard Victor Dutrow was born December 30, 1880, in Charlesville, Maryland. He received his medical degree in 1904 from the University of Maryland School of Medicine and was an intern at the University of Maryland Hospital. He was a member of the medical department of the Isthmian Canal Commission and served as chief of the eye, ear, nose and throat clinic, Colon Hospital, Canal Zone.

He was past president of the Montgomery County Medical Society and of the Dayton Academy of Medicine; a vet-

eran of World War II; Life Fellow of the American Academy of Ophthalmology and Otolaryngology; a fellow of the American College of Surgeons. He was associated with the Miami Valley Hospital, where he was formerly chief of staff, St. Elizabeth and Good Samaritan hospitals.

Dr. Dutrow died March 17, in Dayton, Ohio.

HOWARD DANIEL FALLOWS

Howard Daniel Fallows, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, was born in Waucoma, Iowa, May 31, 1884. His primary education was obtained in Waucoma and he was graduated from the College of Medicine, State University of Iowa, Iowa City, Iowa. Postgraduate work was done in Philadelphia, Pennsylvania; Denver, Colorado; and Vienna, Austria. He began practicing medicine in Fredericksburg, Iowa, and was later associated with the Park Hospital Clinic (1920-1933) and St. Joseph's Mercy Hospital (1933-1957).

Dr. Fallows was a member of the American Medical Association, the American Association of Railway Surgeons, and a fellow of the American College of Surgeons.

Dr. Fallows died in Tucson, Arizona, December 27, 1957. He is survived by his wife, Blanche; a daughter, Mrs. Willis O. Patton of Mason City, Iowa; and a son, Colonel Ronald F. Fallows, U.S.A.F., Fallo Church, Virginia.

IVAN FAWCETT

Ivan Fawcett was graduated from the Linsly Military Institute in 1906. At the time of his death, February 4, he was president of the board of this organization. He received his medical degree from the University of Pennsylvania in 1918 and took further specialty training at the Wills Eye Hospital, Philadelphia, and at the New York Eye Infirmary. He was certified by the American Board of Otolaryngology, and in 1913 began practicing his specialty in Wheeling, West Virginia. He belonged to the American Medical Association, and the West Virginia Academy of Ophthalmology; he was a fellow of the American College of Surgeons and for many years was a member of the House of Delegates of the American Medical Association. He was a member of the Ogelbay Institute and at one time served as its president. He was on the staff of the Ohio Valley General Hospital.

Dr. Fawcett is survived by his wife, Florence York Fawcett; three sons, Ivan York Fawcett, Cincinnati, Ohio, John Everett Fawcett of Seattle, Washington, and Dr. R. Alan Fawcett of Wheeling; and nine grandchildren.

ALEXANDER FEHER

Alexander Feher was born October 5, 1908, in Budapest, Hungary. He received his medical degree from the Medizinische Fakultat der Universitat, Vienna, Austria, in 1932. He was a resident in ear, nose and throat for two years at the Polyclinic Hospital, Budapest, and for one year at the Harlem Eye and Ear Hospital, New York. He was certified by the American Board of Otolaryngology; was a Fellow of the American Academy of Ophthalmology; assistant professor of otorhinolaryngology at the New York University College of Medicine and the New York University Post-Graduate Medical School, and assistant professor of clinical otolaryngology at the Yale University School of Medicine. He was on the staff of the Manhattan Eye, Ear and Throat Hospital, New York, and associated with the City Hospital, New York, and the Veterans Administration Hospital, West Haven, Connecticut.

Dr. Feher died September 6, 1957, of coronary thrombosis and arteriosclerosis. He is survived by his widow and a daughter.

EVERETT RUSSELL FERGUSON

Everett Russell Ferguson was graduated from North Carolina Medical College in 1917. Soon after service in World War I, he was certificated by the American Board of Otolaryngology and began practice in Tennessee.

Dr. Ferguson was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association and a fellow of the American College of Surgeons. He died, aged 65, in Cleveland, Tennessee, March 15.

MAXWELL FINEBERG

Maxwell Fineberg was born in Montreal, Quebec, Canada, June 2, 1900. He

received the degrees of Doctor of Medicine and Master of Surgery from McGill University in 1923.

After postgraduate work in Vienna in 1927 and 1928, Dr. Fineberg was certified by the American Board of Otolaryngology. He began practicing in St. Louis where he was associated with the Central Institute for the Deaf, was assistant editor of the Laryngoscope and served on the staff of Jewish Hospital. From 1948 to 1953, he served as head of the Department of Otolaryngology of Jewish Hospital.

Dr. Fineberg was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association. He died in St. Louis, March 8, survived by his wife, Jeanne Marx and sons, Mark, Daniel and Richard.

JERRY M. GARBER

Jerry M. Garber, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Mansfield, Ohio, September 30, 1957. He was born February 12, 1872, in Bellville, Ohio.

After receiving his degree of Doctor of Medicine from Western Reserve Medical School in 1895, Dr. Garber did postgraduate work at the Philadelphia Polyclinic Hospital, Wills Eye Hospital, Jefferson College, University of Pennsylvania, the Central London Throat and Ear Hospital, the Royal London Ophthalmic Hospital, the University of Vienna and the Polyclinic Hospital of Vienna.

He practiced opthalmology and otolaryngology in Mansfield where he was on the staff of Mansfield General Hospital. Dr. Garber was a fellow of the American College of Surgeons, a member of the American Medical Association and the American Medical Association of Vienna. He was honored with the 50 Year Award of the Ohio State Medical Association.

Dr. Garber is survived by his wife, a son, Dr. C. Zent Garber of New York, and a daughter.

CHARLES HOWARD GORDON

Charles Howard Gordon was born July 7, 1894, in Ashland, New Hampshire. He was graduated from the Medical School of Maine in 1919, and certified a specialist by the American Board of Otolaryngology. In 1923, Dr. Gordon began the practice of medicine in Portland, Maine.

Dr. Gordon was a consultant at the Webber Hospital in Biddeford, Maine, on the staff of the Maine General Hospital, and chief of the ear, nose and throat service at the Maine Eye and Ear Infirmary.

He was a past president of the Cumberland County Medical Society, a Life Fellow of the American Academy of Ophthalmology and the American College of Surgeons, and a member of the American Medical Association.

Before his death, July 28, Dr. Gordon enjoyed hunting, fishing and golf. His widow and daughter survive him.

THOMAS FRANCIS GOWEN

Thomas Francis Gowen, born July 28, 1905, in Philadelphia, Pennsylvania, retired June, 1951, from the U. S. Navy with the rank of Commander. He received his medical degree from Temple University School of Medicine, Philadelphia, in 1929. He did postgraduate work in laryngology at Harvard University in 1937 and at one time was an instructor in the Graduate School of the University of Pennsylvania. He served in the U. S. Navy during World War II and transferred to the regular Navy in 1948. He was certified by the American Board of Otolaryngology and following his retirement from the Navy he opened his practice in otolaryngology in Corona Del Mar, California. Dr. Gowen was a Fellow of the American Academy of Ophthalmology and Otolaryngology.

He died October 19, 1957, of coronary disease, and is survived by his widow, Isabelle M. Gowen, and two daughters, Mrs. Frances A. Kennedy of Huntington Beach, California, and Miss Mabel Gowen, Garden Grove, California.

EDWIN ALBERT GRIFFIN

Edwin Albert Griffin, a specialist certified by the American Board of Ophthalmology, received his medical degree from the University and Bellevue Hospital Medical College, New York, in 1909. He was a past president of the Pan American Medical Association; served as vice president of the Kings County Medical Society; was a Fellow of the American Academy of Ophthalmology and Otolaryngology; a member of the American

Laryngological, Rhinological and Otological Society; American Otorhinologic Society for Plastic Surgery, and American College of Allergists; a fellow of the American College of Surgeons and the International College of Surgeons; he served on the faculty of Long Island College of Medicine, and was associated with Brooklyn Eye and Ear, Samaritan and Methodist hospitals.

Dr. Griffin was born in New York, May 25, 1887. He died December 29, 1957, in Brooklyn, New York, of cardiac failure. He is survived by his widow, Mrs. Ethel F. Griffin; a son, Dr. E. Harrison Griffin; and two grandsons.

MILUS LIDDELL GUNN

Dr. Milus Liddell Gunn, a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, was born in Woodland, Texas, November 29, 1895. He was graduated from the University of Louisville School of Medicine, Louisville, Kentucky, and took additional specialty training at the New York Post-Graduate School and Hospital, New York Eye and Ear Infirmary, New York Polyclinic Medical School, University of Vienna, Vienna, Austria, University of Berlin, Berlin, Germany and studied eye surgery under Sir Henry Howard, Shikapur, India. He was certified by the American Board of Otolaryngology.

He was consulting ophthalmologist for the Louisville and Nashville Railroad Companies; he served on the staffs of the Harlan Memorial and Harlan hospitals. He was a fellow of the International College of Surgeons and the American College of Surgeons; he belonged to the Harlan County Medical Society, Kentucky State Medical Association and the Southern Medical Association.

Dr. Gunn died April 3, in Pittsburgh, Pennsylvania. Surviving are his widow; a son, Dr. William Hughes Gunn; a daughter, Martha Gunn Morgan; and four grandchildren.

HARRY H. HAGGART

Harry H. Haggart received his Bachelor of Arts degree from the Fargo College, Fargo, North Dakota, and his Doctor of Medicine degree from The Johns Hopkins University School of Medicine, Baltimore, Maryland, in 1921. He did postgraduate work in New York and Detroit; began his medical practice in 1928, and was later certified by the American Board of Otolaryngology. He belonged to the American Society of Plastic and Reconstructive Surgery; American Rhinological Society; American Medical Association; and the American Academy of Ophthalmology and Otolaryngology. He was associated with Children's and Christ hospitals in Cincinnati, Ohio.

Dr. Haggart was born June 20, 1892, and died in Cincinnati July 14. He is survived by his widow, Jessie Hoslin Haggart; two daughters, Mrs. George C. Kaiser, and Mrs. Eric B. Yeiser; and six grandchildren.

ALFRED WILLIAM HASKELL

Alfred William Haskell was born in Portland, Maine, February 8, 1876. He was graduated from Bowdoin Medical School in 1900. During World War I, Dr. Haskell distinguished himself as an eye surgeon in the army hospitals in France, and after the war he was active in the Medical Officers' Reserve Corps. He was a director of the Maine Institution for Blind, a Mason, and a former president of the Past Commanders Association of the Harold T. Andrews Post, American Legion. He maintained memberships in the Maine Medical Association, American Medical Association, Portland Medical Club, Portland Club and the Cumberland Club, and he was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Haskell died in Portland, November 21, 1957 of bronchopneumonia and bronchiectasis. He is survived by a sister, Louise F. Haskell, of Portland.

WILLIAM JACOB HERTZ

William Jacob Hertz received his Doctor of Medicine degree from the Medico-Chirurgical College, Philadelphia, Pennsylvania, in 1898. He did postgraduate work at the Graduate School of the University of Pennsylvania in 1908 and for one month during each of five summers he did postgraduate work in Vienna. He was associated with the Sacred Heart and Allentown hospitals, serving as chief of the eye, ear, nose and throat department of the latter from 1916 to 1946.

He was a past president of the Lehigh County Medical Association, and had

served as its treasurer for twenty-five years from 1925 to 1950. He belonged to the Pennsylvania State Medical Society, the American Medical Association, Pennsylvania Academy of Ophthalmology, American Academy of Ophthalmology and Otolaryngology, and was a fellow of the American College of Surgeons. Dr. Hertz was a diplomate of the American Board of Ophthalmology.

Dr. Hertz was born November 3, 1876, in Allentown, Pennsylvania. He died in Allentown May 4. He is survived by a son, Charles S. Hertz, M.D.; two daughters, Mrs. Isabelle H. Gamble and Mrs. Catharine S. Wagner; and four grandchildren.

JOHN McCHESNEY HOGSHEAD

John McChesney Hogshead, born June 15, 1876, near Middlebrook, Virginia, received his Doctor of Medicine degree from the Virginia Medical College, Richmond. He received further training in his specialty in New York, London, Paris and Vienna. He opened his practice, limited to diseases of the eye, ear, nose and throat, in 1906, in Chattanooga, Tennessee, and served on the staffs of all of the local hospitals in Chattanooga.

Dr. Hogshead died July 13. He is survived by his widow; a daughter, Mrs. Paul L. Tillson; four grandchildren; and a sister, Mrs. W. W. Turner.

JAROSLAV H. HULKA

Jaroslav H. Hulka, aged 64, died June 18, in Long Island, New York. He had received his Doctor of Medicine degree in 1919 from the Czech University of Prague. He practiced otolaryngology in New York for many years. Dr. Hulka was a member of the American Medical Association and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

VESSE REEVES HURST

Vesse Reeves Hurst was born October 5, 1887, in Timpson, Texas. In 1912 he received the degree of Doctor of Medicine from the University of Texas Medical College in Galveston. Dr. Hurst did postgraduate work in EENT in New York before establishing practice in Longview, Texas, in 1914. There Dr. Hurst built one of the first EENT private hospital clinics in Texas. He was a charter member and vice president of the Private Clinics and Hospitals Association of Texas.

Dr. Hurst was a diplomate of the American Board of Ophthalmology and a Life Fellow of the American Academy of Ophthalmology and Otolaryngology as well as a fellow of the American College of Surgeons and past president of the Tri-State Medical Society of Arkansas, Louisiana and Texas. He was an active member of the Southern Medical Association, past president of the Gregg County Medical Society and a member of the American Medical Association.

In his community, Dr. Hurst served as vice president and director of the First National Bank, former president and director of the Longview Saving and Loan and vice president and director of the Kelly Plow Company. He was first president of the Longview Chamber of Commerce.

Dr. Hurst was associated with the Hurst Eye, Ear, Nose and Throat Hospital, the Gregg Memorial Hospital and the Markham Memorial Hospital, where he died December 7, 1957. He is survived by his wife, Genevieve, two daughters, Mrs. Oscar Marchman and Mrs. Blackshear Jameson, and three grandchildren.

CHEVALIER JACKSON

Chevalier Jackson was born in Pittsburgh, Pennsylvania, November 4, 1865, and died August 16, in Philadelphia, Pennsylvania.

He received the degree of Doctor of Medicine from Jefferson Medical College in 1886, and began the practice of medicine, specializing in laryngology, in Pittsburgh the following year. In 1912, he was named professor of laryngology at the University of Pittsburgh, and in 1916 he moved to Philadelphia where he was elected professor of laryngology at Jefferson Medical College.

Dr. Jackson was elected to fill the new chair of bronchoscopy and esophagoscopy created and endowed for him by the Board of Trustees of the University of Pennsylvania, at the request of the Graduate School of Medicine. In 1921, he was elected professor of bronchoscopy and esophagoscopy, a chair created for him by Jefferson Medical College. At his request, Dr. Fielding O. Lewis was elected to fill the chair of laryngology.

He was elected lecturer on bronchoscopy and esophagoscopy at Woman's Medical College of Pennsylvania. Temple

University created a chair of bronchoscopy and esophagoscopy in 1930 and elected Dr. Jackson professor. In the same year he was elected professor emeritus of bronchoscopy and esophagoscopy of the Graduate School of Medicine of the University of Pennsylvania. In 1934, he was elected professor of bronchoscopy and esophagoscopy at the Woman's Medical College.

Dr. Jackson pioneered in the field of bronchoscopy and esophagoscopy and invented many of the instruments used in his work. He was made an honorary member of many foreign and American societies and received many honorary degrees.

Dr. Jackson's academic career was unique in that he held five professorships synchronously, a number of honorary and emeritus professorships, lectureships and membership in a number of American and foreign organizations. He was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1904.

Textbooks by Dr. Jackson have been translated into French, Italian, Spanish and Japanese. His contribution to medical literature includes hundreds of articles.

Dr. Jackson is survived by his son, Dr. Chevalier L. Jackson.

HOBART CLEMENS JOHNSON

Hobart Clemens Johnson was born in Olsburg, Kansas, March 11, 1897. He received his Bachelor of Arts degree from Gustavus Adolphus College in St. Peter, Minnesota, and, in 1926, he was awarded the degree of Doctor of Medicine from the University of Minnesota. He received the Diploma in Tropical Medicine and Hygiene in England in 1928.

Dr. Johnson served as a medical missionary in Tanganyika Territory in East Africa for ten years before setting up practice in Mankato, Minnesota. He was on the staffs of Immanuel and St. Joseph's hospitals in Mankato. Dr. Johnson became a diplomate of the American Board of Ophthalmology in 1944.

In addition to the Academy, Dr. Johnson belonged to the Association for Research in Ophthalmology, the Royal Society of Tropical Medicine and Hygiene (England), the World Medical Association, the Minnesota Academy of Science and the American Medical Association. He was past president and a member of the council of the Minnesota Academy of Ophthalmology and a senior member of the Pan-American Association of Ophthalmology.

Dr. Johnson was a member of the American Scandinavian Foundation and a life member of the Swedish Colonial Society. He also belonged to the American Swedish Institute and the Swedish Pioneer Historical Society. In 1957 he received the Greater Gustavus Award, an alumni citation from Gustavus Adolphus College.

Dr. Johnson died July 23, 1957, in Duluth, Minnesota. He is survived by his wife, Ruth, two daughters, Dorothy and Marjorie (Mrs. Russell Knutson), and one son, Dr. Wendell Johnson.

CECIL FREDERICK JOHNSTON

Cecil Frederick Johnston was graduated from the Medical College of Virginia in 1929 and certificated by the American Board of Otolaryngology. He began practicing medicine in Princeton, West Virginia, in 1931.

Eventually, Dr. Johnston settled in Bluefield, West Virginia. He served as head of the otolaryngology department of Bluefield Sanitarium and was an active member of the hospital staff.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the West Virginia Society of Ophthalmology and Otolaryngology and the American Medical Association.

Dr. Johnston supported the Y.M.C.A. and gave financial aid to young people who wished to continue their educations. He was interested in fishing. His colleagues will remember his practical jokes. He died July 21, in Bluefield, aged 54, of coronary occlusion. His wife survives him.

ALEXANDER SPENCER KAUFMAN

Alexander Spencer Kaufman was born October 21, 1883, in Quincy, Illinois. He received his Doctor of Medicine degree in 1904 from the Jefferson Medical College, Philadelphia, Pennsylvania, and was certified by the American Board of Otolaryngology after having done postgraduate study in bronchoscopy and fenestra-

tion surgery. He was associated with the Jewish Hospital in Philadelphia, serving as chief of the ear, nose and throat section from 1914 until 1951. He was consultant for the Delaware County and Mercy Douglas hospitals in Philadelphia, and was clinical professor of ear, nose and throat at Jefferson Medical College from 1911 until his death.

Dr. Kaufman belonged to the Philadelphia County Medical Society, Medical Society of the State of Pennsylvania, Medical Club of Philadelphia, American Medical Association, Fenestration Study Group; he was a fellow of the American College of Surgeons and a Life Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a charter member of Phi Chi fraternity, Jefferson Hospital, and served on the board of trustees of the National Agricultural College, Doylestown, Pennsylvania, and the Association for Jewish Children, Philadelphia.

He died March 30, in Philadelphia. Surviving are his widow, Florence; a daughter, Mrs. Dorothy (J. C.) Goldsmith; and two grandchildren.

FRANK CONRAD KEIL

Frank Conrad Keil was born in Manhattan July 13, 1884. He graduated from Cornell University Medical College in 1907 and then completed a two-year rotating internship at the Methodist Episcopal Hospital. Dr. Keil first engaged in general practice, but soon became interested in the eye, ear, nose and throat and eventually was certified by the American Board of Ophthalmology and the American Board of Otolaryngology. He received additional specialty training at the Harlem Eye and Ear Hospital and for a time was associated with St. Luke's and the New York Eye and Ear Hospital as well as the Out-Patient Department at Cornell.

In 1931 he spent almost a year in postgraduate study in ophthalmology in Vienna and thereafter restricted himself to this specialty. He joined the staff of the Manhattan Eye, Ear and Throat Hospital, later becoming the surgeon-director. At the time of his death he was consultant in ophthalmology at the Manhattan Hospital, at St. Clare's and Pilgrim State hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Alumni Society of Interns of the Methodist Episcopal Hospital, The Medical Strollers, The West Side Clinical Society, the American Medical Association and the Medical and Surgical Club. In the New York Academy of Medicine he was chairman of the eye section from 1943 to 1944, and he was president of the New York Ophthalmological Society in 1951.

Dr. Keil died suddenly October 15, 1957. He is survived by his widow; a son, Dr. Frank C. Keil, Jr.; and two sisters.

PETER AUGUSTUS KEIL

Peter Augustus Keil was graduated from Long Island College Hospital in 1902 with a Doctor of Medicine degree. He was certificated by the American Board of Otolaryngology in 1928.

Dr. Keil practiced medicine in Brooklyn, New York, for many years and was associated with the Lutheran Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and the American College of Surgeons.

Dr. Keil served as director of the Nassau Savings and Loan Association. He died October 13, aged 78, of acute monocytic leukemia.

LOWELL WALDO KING

Lowell Waldo King of Salem, Ohio, was graduated from Western Reserve University School of Medicine in 1918. He was a diplomate of the American Board of Otolaryngology.

Dr. King served on the staffs of the Central Clinic and Hospital and the Salem City Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association. He died June 29, aged 66.

HENRY KIRKPATRICK

Henry Kirkpatrick, an Honorary Fellow of the American Academy of Ophthalmology and Otolaryngology, died May 11, at his home at Yateley, Hampshire, England. He was 86.

Lt. Col. Kirkpatrick served as an ophthalmologist in the Indian Medical Service for many years; he made a great contribution to the advancement of ophthalmology in Southern India. He acted as

superintendent of the Government Ophthalmic Hospital in Madras and served as professor of ophthalmology.

When he returned to London, Lt. Col. Kirkpatrick opened a consulting practice. At this time, he wrote several books and served as ophthalmic surgeon to the Tropical Diseases Hospital. He also lectured in ophthalmology at the London School of Hygiene and Tropical Medicine.

Lt. Col. Kirkpatrick received his education at Marlborough College and Trinity College, Dublin. He was a member of the Ophthalmological Society of the United Kingdom. Lt. Col. Kirkpatrick is survived by his wife and two daughters.

ALAN DeGRIEF KNISLEY

Alan DeGrief Knisley was born in New Philadelphia, Ohio, February 22, 1881. He attended the University of Louisville, receiving his Doctor of Medicine degree in 1906, after which he did postgraduate work at the New York Post-Graduate School and Hospital. He began his medical practice in Lima, Ohio, serving on the staffs of the St. Rita's and Lima Memorial hospitals.

Dr. Knisley was a member of the American Medical Association; American Laryngological, Rhinological and Otolaryngological Society and the American Academy of Ophthalmology and Otolaryngology. He was on active duty in the medical corps of the United States Army during World Wars I and II, retiring with the rank of lieutenant colonel.

He died August 31, 1957, in Toledo, Ohio. Surviving are a daughter, a son and six grandchildren.

HEINRICH FRANZ GUNTHER KOBRAK

Heinrich Franz Gunther Kobrak was born March 19, 1905, in Berlin, Germany. He received his medical degree from the Friedrich-Wilhelms-Universitat Medizinische Fakultat, Berlin, Prussia, Germany, in 1928, and a Doctor of Philosophy degree from the University of Chicago in 1937. Dr. Kobrak was an assistant professor in otolaryngology at the University of Chicago from 1946 to 1954 and assistant professor in industrial otorhinolaryngology at Wayne University College of Medicine, Detroit, from 1954 to 1957. He was certified by the American Board of Otolaryngology and was a Fellow of the American Academy of Ophthalmology and Otolaryngology.

He belonged to the American Otological Society; American Laryngological, Rhinological and Otological Society; Acoustical Society of America; American Physiological Society; American Association for the Advancement of Science; and the American Medical Association. He was awarded honorary memberships in five organizations, two of which are in South America.

Dr. Kobrak was president of the Michigan State Acoustical Society in 1957. He delivered the Tilderquist Memorial Lecture in Duluth, Minnesota, 1951; the Sigma Xi Lecture at Columbia University, New York, in 1952; the Herbert Bickett Memorial Lecture, McGill University, Quebec, Canada, 1954. He received first prize of surgical films at the International Film Festival for Biological and Medical Films in Turin, Italy, June, 1952, and the Award of Merit of the American Academy of Ophthalmology and Otolaryngology for distinguished services in the educational programs of that society, 1957.

Dr. Kobrak was deeply interested in animal husbandry and operated a dairy farm on which the family made its home. He also enjoyed chamber music and played the viola in a string quartet.

Dr. Kobrak died in Clarkston, Michigan, October 9, 1957, of cerebral vascular accident while preparing to leave for the meeting of the American Otological Society in Chicago, where he was to receive the Award of Merit for outstanding achievement in his field. He is survived by his widow, Jessie Mae Kobrak; a daughter, Gertrude Ann; a son, Donald Walter; and two brothers.

HUGH ALVA ROSS KUHN

Hugh Alva Ross Kuhn was born November 16, 1895 in Lore City, Ohio. He was graduated from the University of Cincinnati Medical College after which he interned at St. Mary's Hospital in Cincinnati; he then served as a resident at the Cincinnati General Hospital. He had further training at the University of Bordeaux, France; University of Vienna, Austria; New York Eye and Ear; Bellevue Medical College; Los Angeles Study Club; Hansel Foundation; and with Dr. Howard House, Los Angeles, California. In 1928, he was granted certificates by

the American Board of Ophthalmology and the American Board of Otolaryngology. His hospital associations were with St. Margaret's Hospital, Hammond, Indiana; St. Catherine's Hospital, East Chicago, Indiana; Our Lady of Mercy Hospital, Dyer, Indiana; and he was consultant at South Chicago Commuity Hospital.

Dr. Kuhn was active and served as an officer in many medical and nonmedical organizations. He was past president of the American Society of Ophthalmologic and Otolaryngologic Allergy and of the Indiana Academy of Ophthalmology and Otolaryngology, former vice president of the American Society of Ophthalmologic and Otolaryngologic Allergy and of the American College of Allergists; secretary of the American Medical Association, Section of Otolaryngology for five years; program chairman for the American College of Allergists, Ear, Nose and Throat Section in 1954 and 1957, Indiana Academy of Ophthalmology and Otolaryngology in 1957, Hansel Foundation in 1957 and the American Academy of Ophthalmology and Otolaryngology in 1957.

Dr. Kuhn died August 17, in Hammond, Indiana. His wife, Dr. Hedwig S. Kuhn, and two sons, both physicians, survive him.

FRITZ LASSEN

Dr. Fritz Lassen, a 1903 graduate of the Ludwig-Maximilians-Universitat Medizinische Fakultat, Munchen, Bavaria, Germany, came to the United States in 1907 and was certified by the American Board of Otolaryngology. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, a member of the American Medical Association and a co-founder of the Pueblo (Colorado) Clinic and the Parkview Hospital, in Pueblo.

Dr. Lassen died in Pueblo June 30, of coronary occlusion. He is survived by his widow, Mrs. Margaret O'Hare Lassen.

RICHARD ORMOND LEAVENWORTH

Dr. Richard Ormond Leavenworth, a 1914 graduate of the University of Minnesota School of Medicine, was a specialist certified by the American Board of Ophthalmology. He did postgraduate work at Cornell University, Washington University, Illinois Charitable, University of Nancy and University of Vienna, Austria. He became a clinical instructor of ophthalmology and otolaryngology at the University of Minnesota and was affiliated with Ancker, Children's, St. Joseph's and St. Luke's hospitals. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons, and belonged to the American Medical Association and the Minnesota Academy of Ophthalmology and Otolaryngology. During World War I he served as an army surgeon in France.

In his leisure, Dr. Leavenworth enjoyed sailing, duck hunting, curling, and photography. He was also interested in astronomy.

Dr. Leavenworth died January 29, in St. Paul, Minnesota, of pulmonary embolism and congestive heart failure. Surviving Dr. Leavenworth are his widow, Geneva H.; two daughters, Mrs. E. A. Post, St. Paul, and Mrs. Harrison H. Cochran, Jr., San Gabriel, California; a son, Richard O. Leavenworth, M.D., and a sister, Mrs. Burt Newkirk, Schenectady, New York.

ALBERT NAPOLEON LEMOINE, SR.

Albert N. Lemoine, Sr., a Life Fellow of the Academy, was born October 13, 1884, in Concordia, Kansas. He received his preliminary degree from the University of Kansas and his medical degree from the University of Kansas School of Medicine. His postgraduate work in ophthalmology was done at Harvard Medical School and the Massachusetts Eye and Ear Infirmary.

Dr. Lemoine established his practice in Kansas City, Missouri, in 1923, and lived there until he died August 30, 1957. He served on the staffs of Menorah Medical Center, Research, St. Joseph, Children's Mercy, and Kansas City General hospitals.

A Fellow of the Academy since 1920, Dr. Lemoine was given an Honor Award in 1948. He was also a member of the American Ophthalmological Society and the Association for Research in Ophthalmology, a fellow of the American College of Surgeons, a member of the American Medical Association, and past president of the Kansas City Society of Oph-

thalmology and Otolaryngology and the Jackson County Medical Society.

He is survived by his wife, Myrle, three daughters and one son, Albert N. Lemoine, Jr., M.D., also a Fellow of the Academy.

WAYLAND HOYT MALOY

Dr. Wayland Hoyt Maloy was a specialist certified by the American Board of Ophthalmology. He was born December 7, 1896, in Redding, Iowa. He received his Doctor of Medicine degree in 1925 from the University of Iowa, Iowa City, and continued at this institution for further study in diseases of the eye, ear, nose and throat. He began his practice in Council Bluffs, Iowa, in 1931 and in Shenandoah in 1932. He served on the faculty of the University of Iowa School of Medicine and was affiliated with the Hand Memorial Hospital, Shenandoah. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and past president of the Page County Medical Society.

Dr. Maloy died December 26, 1957, in Shenandoah of carcinoma of the lung. He is survived by his widow, Jean T. Maloy; a son, John T. Maloy; his mother, Mrs. J. T. Maloy, and a sister, Mrs. Lillian Gilleland.

JOHN HOLLIS MATHESON

John Hollis Matheson, born October 10, 1904 at Ireton, Iowa, attended Drake University in Des Moines, Iowa. He received his Bachelor of Arts degree from Drake in 1924 and then transferred to the University of Iowa School of Medicine from which he was graduated in 1930. He interned at the University of Wisconsin and returned to the University of Iowa for further study in neurology and ophthalmology. He began his medical practice in June, 1935, in Des Moines where he served on the staffs of the Iowa Methodist, Iowa Lutheran and Mercy hospitals.

Dr. Matheson was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and belonged to the American Medical Association and the Iowa and Des Moines Academies of Ophthalmology and Otolaryngology. He was also a member of the Des Moines Civic Music Association. He served in the U. S. Navy from August 1944, to June 1946.

Dr. Matheson died March 11, in Des Moines. He is survived by his wife, Helen; two sons, John, 15, and Steven, 13, and a brother, George.

WILLIS LESLIE McCONKIE

Following an illness of five months, Dr. Willis Leslie McConkie died August 14, 1957, at St. Anthony Hospital, Carroll, Iowa. He had practiced in Carroll since 1920 after having completed an internship at the University Hospitals in Iowa City. Dr. McConkie was graduated from the University Hospital School of Medicine, Iowa City, in 1919.

Dr. McConkie was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He had served as president of the Carroll County Medical Society and was on the staff of St. Anthony's Hospital in Carroll.

He is survived by his widow, the former Edna E. Johnson; one daughter, Mrs. B. V. (Phyllis) McKinney, Northridge, California; one son, Robert L. McConkie, Jefferson; and three grandchildren.

JOSEPH LETELLE McCOOL

Joseph Letelle McCool, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died February 12, in Monrovia, California, aged 78.

Dr. McCool was graduated from the University of Pennsylvania Department of Medicine in 1900. He was a specialist certified by the American Board of Ophthalmology. Dr. McCool was a former faculty member of the University of California School of Medicine. He was also an associate member of the American Medical Association.

JAMES HAROLD MENDEL

James Harold Mendel died at his home in Coral Gables, Florida on February 5, after a long illness. He was born August 13, 1894, in Wheeling, West Virginia; received his degree in medicine from Jefferson Medical College, Philadelphia, in 1918; trained in otolaryngology under Dr. S. McCoen Smith, and was later certified by the American Board of Otolaryngology.

Following service in the United States Navy during World War I he practiced otorhinolaryngology in Germantown, Pennsylvania. He taught neu-

roanatomy at Jefferson Medical School for 18 years. In 1935 he was awarded the Bronze Medal for his exhibit on ear drums at the meeting of the American Medical Association in Atlantic City. He won similar awards for this exhibit from the Southern Medical Association and the Florida Medical Association.

He moved to Miami in 1936 and practiced his specialty. For 10 years he served in the ear, nose and throat department of Jackson Hospital and was later a clinical associate professor of otorhinolaryngology when the University of Miami School of Medicine first opened. He was Selective Service Consultant during World War II after which he helped organize the Greater Miami Eye, Ear, Nose and Throat Society. He was affiliated at various times with all hospitals in the greater Miami area, later limiting his work to the Doctors' Hospital. In 1956 he moved his offices to Coral Gables.

Dr. Mendel belonged to the American Medical Association, Southern Medical Association and was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Mendel is survived by his wife, Kathleen; a stepdaughter, Sandra Lynn Matter; a daughter, Mrs. Dorothy Stanyon, New York, and a son, Dr. James H. Mendel, Jr. of South Miami.

FREDERICK WILLIAM MERRIFIELD

Frederick William Merrifield was born in Plymouth, England, August 4, 1887. He was graduated from Northwestern University Dental School in Chicago, Illinois, in 1916 and Northwestern University Medical School in 1931. Dr. Merrifield was professor of oral surgery emeritus at Northwestern University Dental School; served as chairman of the executive committee of the Northwestern University Cleft Lip and Palate Institute.

He was a specialist certified by the American Board of Plastic Surgery; a member of the American Association of Plastic Surgery and a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons.

Dr. Merrifield had been associated with the Evanston (Illinois) Hospital, Passavant Memorial and Children's Memorial hospitals. He died January 6, of uremia and chronic pyelonephritis.

H. EDWARD MILLER

H. Edward Miller was born February 1, 1878, in St. Louis, Missouri. He was graduated from Washington University School of Medicine in 1902 after which followed a medical career of over 50 years. He was former chief of the department of otolaryngology at Washington University School of Medicine, surgeon-in-charge of the Old O'Fallon Dispensary, and assistant laryngologist at Barnes Hospital and he served on the visiting staff of the City Hospital. He was also consultant to the United States Department of Health.

Dr. Miller was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a senior fellow of the American Laryngological, Rhinological and Otological Society, and he belonged to the St. Louis Medical Society.

He died on August 3, 1957, in St. Louis. Surviving Dr. Miller are two daughters, Mrs. John Burt, Pasadena, California, and Mrs. Theodore S. Cleveland, St. Louis; and two sons, the Rev. Edward O. Miller, New York, and Robert O. Miller, Manistee, Michigan.

HAROLD REXFORD MILLER

Harold Rexford Miller, New York, received his Doctor of Medicine degree from the Toronto Faculty of Medicine, Toronto, Ontario, in 1923. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a diplomate of the American Board of Ophthalmology, and was associated with the New York Eye and Ear Infirmary.

He died at St. Clare's Hospital on December 9, 1957, aged 61. He is survived by his widow, Edna; his father, Robert F. Miller, Cayuga, Ontario; two brothers and two sisters.

HARRY F. MINOR

Harry F. Minor was born in 1880; he was graduated from Vanderbilt University School of Medicine in 1903. Dr. Minor was a diplomate of the American Board of Otolaryngology and practiced ophthalmology and otolaryngology in Memphis, Tennessee, for many years. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association.

WILLIAM HENRY MINTON

William Henry Minton was graduated from Washington University School of Medicine, St. Louis, Missouri, in 1905. He was a diplomate of the American Board of Ophthalmology.

Dr. Minton was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons and a member of the American Medical Association. He was past president of the Buchanan County, Missouri, Medical Society, and of the St. Joseph, Missouri, chapter of the American College of Surgeons.

Dr. Minton died in St. Joseph November 2, 1957, aged 75, of cerebrovascular thrombosis.

WILLIAM MITHOEFER

William Mithoefer, an 1897 graduate of the Medical College of Ohio, opened his medical practice in Cincinnati, Ohio, November 1, 1899, after having completed postgraduate work in Vienna, Austria; Strassburg, Munich and Berlin, Germany; Bern, Switzerland and New York. He served as an instructor at the Medical College, University of Cincinnati, (anatomy, 1900-1904, laryngology, 1905-1918), as laryngologist at Christ Hospital and was at one time on the staff of Good Samaritan Hospital and the Tuberculosis Sanitarium. He conducted the 30-bed Mithoefer Hospital for only ear, nose and throat patients from 1924 to 1930.

He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, belonged to the American Medical Association; Cincinnati Otolaryngological Society; American Otological Society; American Rhinological, Otological and Laryngological Society, and the Cincinnati Academy of Medicine, serving as president of this organization for the year 1929-1930. He was an honorary member of the Kansas City Otolaryngological Society.

Dr. Mithoefer was born November 1, 1875, in Cincinnati. He died there May 26, of acute hemorrhage pancreatitis with embolism of the mesentery. He is survived by his widow, Florence Pusey Mithoefer; three sons, William C. Mithoefer, Cincinnati, Dr. James and Dr. John Mithoefer, Copperstown, New York; and three daughters, Mrs. Jean Verral, New York, Mrs. Martha Brodhead, Cincinnati, and Mrs. Florence Keeler.

WILLIAM F. MOLT

Dr. William F. Molt died at his home in Indianapolis, Indiana, January 12. During his 56 years of medical practice he served on the teaching staff of the Indiana University School of Medicine, becoming professor emeritus in 1952. He had studied bronchoesophagology under Chevalier Jackson, M.D., and later established the bronchoesophagological clinic at the Indianapolis General Hospital, acting as its chief for many years.

Dr. Molt was graduated from the Physio-Medical College of Indiana in 1897. He also attended Toledo Medical College and later did postgraduate work at New York Polytechnic and New York Lying-in hospitals; Manhattan Eye, Ear, Nose and Throat Clinic; Chicago Polytechnic, and at Indiana University. He was a senior member of the Indianapolis Medical Society and belonged to the American Medical Association. He was a Fifty Year Club member of I.S.M.A.; a diplomate of the American Board of Otolaryngology; a Life Fellow of the American Academy of Ophthalmology and Otolaryngology and a senior member of the American Broncho-Esophagological Association.

Dr. Molt is survived by his widow.

DANIEL CAMERON MONTGOMERY

Daniel Cameron Montgomery was born January 6, 1885, in Greenville, Mississippi. He died in Greenville May 29. He attended Vanderbilt University, Nashville, Tennessee, receiving his Bachelor of Arts degree in 1906. After being graduated from Jefferson Medical College, Philadelphia, in 1910, he was a resident at the Methodist Episcopal Hospital in Philadelphia; he then returned to Greenville and began his medical practice in December 1914. He served there on the staffs of General and King's Daughters hospitals.

Dr. Montgomery was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, and a member of the American Laryngological, Rhinological and Otological Society, Southern Medical Association, American

Medical Association; he was past president of the Louisiana-Mississippi Ophthalmological and Otolaryngological Society and past vice president of the Mississippi State Medical Association. He belonged to the Rotary Club and was chairman of the board and past president of the First National Bank as well as a former member of the Board of Education in Greenville. Dr. Montgomery was a well known magician.

He is survived by his widow, Ruth Atterbury, and three sons, Daniel C., Jr., M.D., John A., and William D.

DAVID JACOB MORGENSTERN

David Jacob Morgenstern was graduated from Yale University School of Medicine in 1926. He was a diplomate of the American Board of Otolaryngology and limited his practice to ear, nose, throat and tear sac surgery.

He was a fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association. He died February 12, aged 61, of coronary thrombosis.

EDMONDE DE WITT NEER

Edmonde De Witt Neer, born February 12, 1879, received his Doctor of Medicine degree from Western Reserve University Medical Department, Cleveland, Ohio, in 1907. After further study in Vienna and New York he was certified by the American Board of Otolaryngology. He began his practice in general surgery in Cleveland, moved to New York in 1920 and limited his practice to diseases of the ear, nose and throat. He served on the staffs of the New York and Manhattan Eye and Ear hospitals. Dr. Neer was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons; he belonged to the West Side Clinical Society, Medical Strollers, and the Medical and Surgical Society.

Dr. Neer died December 6, 1957, in New York. Mrs. Neer survives her husband.

FRITZ NELSON

Fritz Nelson was born August 23, 1890, Berlin, Germany. He received his Doctor of Medicine degree from the University of Kiel in 1916. He practiced medicine in Rostock, Germany, for several years before coming to the United States.

Dr. Nelson had been an eye specialist in Colorado Springs, Colorado, since 1937. He was a staff member of the Colorado Medical School, on the Colorado Committee for the National Society for Prevention of Blindness, Inc., and a member of the Colorado Ophthalmological Society. He was also a member of the Pan-American Society of Ophthalmology and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Interests other than medicine included ranching, painting and zoology. Dr. Nelson died January 2, aged 67. He is survived by his wife and four children.

MAX NISSELSON

Max Nisselson was born in 1874 and was graduated from Columbia University College of Physicians and Surgeons in 1905. He practiced ophthalmology and otolaryngology in New York, New York, for many years.

Dr. Nisselson was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology and an associate member of the American Medical Association. He died May 12.

CARL MAGNUS OBERG

Carl Magnus Oberg was born July 14, 1876. He attended Latin School in Stockholm, Sweden, and received his degree of Doctor of Medicine from Hamline University in St. Paul, Minnesota, in 1899. He did postgraduate work in Chicago, Illinois, and Vienna, Austria.

Dr. Oberg was associated with the Lutheran Deaconess Hospital of Minneapolis. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the Minnesota Academy of Ophthalmology and Otolaryngology, and a life member of the American Medical Association.

He was a member of the Swedish Society, the American Swedish Institute, the Arcana Blue Lodge, Oneida Chapter and Davies K. T. Zurah Shrine.

Dr. Oberg died on February 6, in Minneapolis. He is survived by his daughter, Miss Margaret Oberg.

DENNIS FRANCIS O'CONNOR

Dennis Francis O'Connor was born in Worcester, Massachusetts, June 11, 1872.

He attended the Holy Cross Preparatory School and College in Worcester, and received his degree of Doctor of Medicine from College of Physicians and Surgeons in Baltimore, Maryland, in 1898. He did postgraduate work at the University of Innsbruk, University of Vienna and the University of Berlin. He received two honorary degrees of Doctor of Laws, one from Holy Cross College in 1925 and the second from Seton Hall University in 1955.

Dr. O'Connor was associated with St. Vincent's Hospital, Worcester City Hospital and Memorial Hospital in Worcester. He was consultant oculist and aurist at Lancaster Industrial School for Girls at Lancaster, Massachusetts, and an instructor at Clark University in the School of Child Study (Department of Psychology) in Worcester.

Military duty interrupted his medical practice in Worcester in 1917. He resumed his civilian practice of medicine in Newark, New Jersey, in 1919. He was associated with St. Mary's Hospital in Orange and St. Michael's Hospital in Newark.

Among Dr. O'Connor's professional affiliations were memberships in the American Academy of Ophthalmology and Otolaryngology and various state and local medical societies. He was a fellow in the American College of Surgeons.

He had many interests and hobbies. Chief among these were traveling, studying and teaching. He spent many of his summers and vacations touring various countries.

Dr. O'Connor died November 2, 1957, in South Orange. He is survived by his wife, Loretta Radel O'Connor.

HENRY BOYLAN ORTON

Henry Boylan Orton was born in Newark, New Jersey, February 6, 1886. He attended Nazareth Hall Military Academy and Dwight School in New York, New York, and received his degree of Doctor of Medicine from Jefferson Medical College, Philadelphia, Pennsylvania, in 1908.

Dr. Orton held many hospital and teaching appointments in the New York area.

Doctor Orton was past president of the American Laryngological Association, and of the American Bronchoscopic Society. He was a fellow and ex-governor of the American College of Surgeons and a fellow of the American College of Chest Physicians. He was a Fellow and past vice president of the American Academy of Ophthalmology and Otolaryngology; a member of the American Laryngological, Rhinological and Otological Society; American Board of Otolaryngology; and was chairman of the Section on Laryngology, Otology and Rhinology of the American Medical Association from 1948 to 1949. He belonged to various local and state medical societies.

He did postgraduate work in New York, Philadelphia, London, Bordeaux, and Boston. He was a captain in the New Jersey National Guard; captain in the Medical Corps in the Mexican Border Service in 1916; and captain in the Medical Corps with the American Expeditionary Forces in World War I. He served with the War Department in the Administration of the Selective Training and Service Act of 1940.

Dr. Orton received the Casselberry and Newcomb awards from the American Laryngological Association; the Honor Key award from the American Academy of Ophthalmology and Otolaryngology; and the Edward J. Ill award was presented to him by the Academy of Medicine of Northern New Jersey in 1953.

Dr. Orton died September 1, 1957, in Newark. He is survived by his wife, Gladys Nicol Orton, and a daughter, Elizabeth Boylan Orton.

LOUIS OSTROM

Louis Ostrom was born in Smaland, Sweden, May 1, 1874. He attended Augustana College in Rock Island, Illinois, and received his degree of Doctor of Medicine from Missouri Medical College in St. Louis in 1899. He did postgraduate work in New York, St. Louis, Chicago and Paris.

Dr. Ostrom was associated with the Rock Island County Tuberculosis Sanatorium, on the staffs of Moline Public and Lutheran hospitals in Moline and St. Anthony's Hospital in Rock Island. He was a fellow of the American College of Surgeons, a member of the American Laryngological, Rhinological and Otological Society and the American Medical Association. Dr. Ostrom also belonged to the Central Illinois Society of Ophthal-

mology and Otolaryngology, the Chicago Laryngological and Otological Society, the Association of Railroad Surgeons and the Association of Military Surgeons.

Music was an important part of Dr. Ostrom's life. He was director of the Shrine Band for more than 20 years. He organized the Boy Scout Band. His civic interests were many. He was awarded a 50-year pin from the John Ericson Republican League of Illinois.

Dr. Ostrom died August 20, 1957, in Rock Island. He is survived by his wife, Sophia, six sons, thirteen grandchildren and four great-grandchildren.

THEODORE FELIX PAPROCKI

Theodore Felix Paprocki was graduated from Long Island Medical College in 1938. He was a diplomate of the American Board of Ophthalmology and practiced his specialty in Ogdensburg, New York, until his death April 30.

Dr. Paprocki was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons and a member of the American Medical Association. He was associated with the A. Barton Hepburn and St. Lawrence State hospitals. He is survived by his wife and three children.

SIR JOHN HERBERT PARSONS

Sir John Herbert Parsons died October 7, 1957, aged 89, in London. He was author of "Diseases of the Eye" and "The Pathology of the Eye." Sir John was president of the Ophthalmological Society of the United Kingdom in 1925 and of the Section of Ophthalmology of the British Medical Association in 1923 and 1932. He was the only ophthalmologist to become president of the Royal Society of Medicine; he held that office from 1936 to 1938. Sir John was chairman of the British Journal of Ophthalmology for 22 years.

Sir John was an Honorary Fellow of the American Academy of Ophthalmology and Otolaryngology. He also received the Lucien Howe Medal of the American Ophthalmological Society in 1936.

CLARENCE N. PEELER

Clarence N. Peeler, a Life Fellow of the Academy, was born in Charlotte, North Carolina, June 21, 1879. He received his preliminary education at Wake Forest College and his degree of Doctor of Medicine from North Carolina Medical College.

Dr. Peeler served as professor of anatomy for North Carolina Medical College as well as director of clinics. He was the first chief of the department of otolaryngology of the Charlotte Memorial Hospital and on the staffs of Presbyterian Hospital and Charlotte Sanatorium.

He was a member of the American Medical Association, a fellow of the American College of Surgeons, and past president of the Mecklenberg County Medical Society.

He died August 28, 1957, survived by two sisters.

JOHN CHANEY PETERSON

John Chaney Peterson, born August 31, 1902, was graduated from the University of Nebraska College of Medicine in 1928. He became a diplomate of the American Board of Ophthalmology in 1935. Dr. Peterson practiced most of his life in Lincoln, Nebraska. He was on the staff of the Lincoln General Hospital.

Dr. Peterson was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association and president of the Nebraska Academy of Ophthalmology and Otolaryngology. He died February 13.

FRANK WILMORE POINDEXTER

Frank Wilmore Poindexter was born in 1887 in Bedford County, Virginia. He was graduated from the Medical College of Virginia in 1913. He began the practice of medicine in Virginia and specialized in otolaryngology. He was a diplomate of the American Board of Otolaryngology.

Dr. Poindexter was a member of the staffs of Riverside and Mary Immaculate hospitals, a consultant at Dixie and Whitaker Memorial hospitals. He was a Fellow and a member of the Board of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, a member of the American Medical Association, the Virginia Society of Ophthalmology and Otolaryngology and the Tidewater Ophthalmological and Otolaryngological Society.

Dr. Poindexter enjoyed his membership in the Masons as well as a day on the golf course. He died in Newport News, Virginia, July 4, of coronary thrombosis. One daughter, two brothers and three sisters survive him.

RALPH ALEX POIRIER

Ralph Alex Poirier was born February 7, 1917, in Forest Lake, Minnesota. He attended St. Thomas College in St. Paul, Minnesota, and received his Doctor of Medicine degree from St. Louis University, St. Louis, Missouri. He studied ophthalmology on a fellowship from the Mayo Clinic, Rochester, Minnesota.

Although he made his home in Birmingham, Michigan, Dr. Poirier practiced his specialty in Detroit, Michigan. He was chief of staff of Mount Carmel Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the Detroit Ophthalmological Society and the American Medical Association.

Dr. Poirier enjoyed both golf and bridge. He died September 28, 1957. His wife, June, and five children survive him.

LAWRENCE TYLER POST

See In Memoriam in the TRANSACTIONS, May-June 1958, following page 504.

ROBERT LINTON RALSTON

Robert Linton Ralston was born in Middlesboro, Kentucky, February 2, 1920. He attended Mount St. Mary's College in Emmitsburg, Maryland, and received his degree of Doctor of Medicine from Georgetown University, Washington, D. C. He did postgraduate work in New Orleans.

Doctor Ralston was associated with Spartanburg General Hospital, Spartanburg, South Carolina; Providence Hospital of Washington, D. C.; and Veterans Administration Hospital of Columbia, South Carolina.

He was a member of the South Carolina Medical Society, Spartanburg Medical Society, Southern Medical Society, South Carolina Society of Ophthalmology and Otolaryngology, and the American Medical Association. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology.

He was an active member of the Sertoma International (Civic Club) of Spartanburg.

Dr. Ralston died April 26, in Spartanburg. He is survived by his wife, Ruth, and four children, Patricia, Katherine, Robert Linton, Jr., and Ruth.

MORRIS MUNICE ROSSMAN

Morris Munice Rossman was born in Brooklyn, New York, December 3, 1909. He attended Syracuse University, Syracuse, New York, and received his degree of Doctor of Medicine from that University. He did postgraduate work at the Syracuse University.

He served on the staffs of University Hospital in New York, New York, the South Nassau Communities Hospital in Oceanside, and the Doctors' Hospital in Freeport, Long Island.

Among Dr. Rossman's professional affiliations were memberships in the state and local medical societies. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and was a specialist certified by the American Board of Otolaryngology.

He was interested in the welfare of his local community and was active in the Exchange Club of Freeport, B'nai Brith, Long Island Zionists Organization, Men's Club of Temple B'nai Israel, and the Sunrise Lodge of the Masonic Order.

Dr. Rossman died February 4, in Freeport, New York. He is survived by his wife, Hortense, and a son, Seth.

MARTIN VAN BUREN RUSSELL

Martin Van Buren Russell was born in Town Creek, Georgia, July 3, 1881. He attended Sewanee Medical School, Atlanta Medical School, and received his degree of Doctor of Medicine from Chattanooga Medical College in 1904. He did postgraduate work at Tulane University.

He served on the staff of the Warner Brown Hospital in El Dorado, Arkansas. Dr. Russell was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and was a member of the American Medical Association, Southern Medical Association, and American Association of Railway Surgeons. He was a fellow of the American College of Surgeons.

Dr. Russell was interested in the program for handicapped children. He was

a member of the Rotary Club, the American Legion, and a member of the Order of the Mystic Shrine. He was a member of the Medical Advisory Board for World War I and also for World War II.

Dr. Russell died February 18, in Hot Springs, Arkansas. He is survived by his wife, Leatha Elna Russell, and a daughter, Mrs. Garth Akridge, and one grandson, Russell Akridge.

LINUS MARTIN RYAN

Linus Martin Ryan was born in Falls City, Nebraska, December 9, 1887. He attended the St. Louis University School of Medicine and received his degree of Doctor of Medicine from that University. He did some postgraduate work at St. Francis Hospital in Pittsburgh, Pennsylvania.

He was affiliated with the St. Louis University School of Medicine, and was on the staff of St. John's Hospital and associated with the St. Mary's Hospital Group in St. Louis. He was a specialist certified by the American Board of Otolaryngology and was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a fellow of the American College of Surgeons. He was a member of the American Medical Association and of the Missouri State Medical Association.

He was a veteran of World War I. Dr. Ryan was interested in community activities and was particularly interested in football and baseball.

Dr. Ryan died September 12, 1957, in St. Louis, Missouri. He is survived by his wife, Corinne Ryan, and one son, Dr. Robert E. Ryan.

FRANCIS S. SCHWARZ, JR.

Francis S. Schwarz, Jr., Shaker Heights, Ohio, died in an automobile accident August 31. He was 37.

Dr. Schwarz was graduated from Cornell University Medical College in 1943 and did postgraduate work at Wayne University. He was certified by the American Board of Ophthalmology in 1951. He practiced his specialty in Cleveland, Ohio, before opening an office in Shaker Heights.

He was on the staffs of St. Vincent Charity and St. Luke's hospitals. He was also affiliated with Curtiss Clinic in Chagrin Falls, Ohio. Dr. Schwarz was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the Cleveland Ophthalmologist Club and the American Medical Association. He is survived by his wife, Ruth, his parents, one brother and two sisters.

ISADORE MARWELL SHAPERO

Isadore Marwell Shapero was born in 1884 in Syracuse, New York. He received his Doctor of Medicine degree from Cornell University in 1907. The University of Vienna gave him an opportunity for postgraduate study. Dr. Shapero was a diplomate of the American Board of Otolaryngology.

He served on the staffs of St. Mary's and Park Avenue hospitals in Rochester, New York. He was an associate member of the American Medical Association and a Life Fellow of the American Academy of Ophthalmology and Otolaryngology. Dr. Shapero died October 27, 1957. Mrs. Rose Shapero survives her husband. A son and daughter are also living.

RELZA NEWTON SHERMAN

Relza Newton Sherman died January 16 in Bradenton Beach, Florida. He was 70. Dr. Sherman retired to Florida in 1951 after practicing in Bay City, Michigan, for many years. He had been associated with Mercy and General hospitals.

Dr. Sherman received his Doctor of Medicine degree in 1913 from the University of Tennessee College of Medicine. He did postgraduate study at the University of Indiana and the University of Michigan. He was a diplomate of both the American Board of Ophthalmology and the American Board of Otolaryngology. Dr. Sherman was an Inactive Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association. His wife, Helen, survives him.

OLGA LAZAROVNA SITCHEVSKA

Olga Lazarovna Sitchevska received her Doctor of Medicine degree from the University of St. Vladimira Faculty of Medicine, Kiev, Russia, in 1918. She was a specialist certified by the American Board of Ophthalmology.

Dr. Sitchevska was on the staffs of the New York Infirmary and the New York Eye and Ear Infirmary. She was a Senior

Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the Association for Research in Ophthalmology and the American Medical Association. She died May 20, aged 62, of coronary thrombosis.

WILLIAM STEINMAN

William Steinman was born in Chicago, Illinois, in 1916. He was graduated from the University of Illinois College of Medicine in 1941; after service in the army, he received postgraduate training at Harvard University and Cook County Hospital of Chicago. After certification by the American Board of Ophthalmology, Dr. Steinman began his practice in Miami, Florida.

He was associated with the Mount Sinai Hospital in Miami Beach, and Mercy and Jackson Memorial hospitals in Miami. He was clinical assistant professor of ophthalmology at the University of Miami School of Medicine in Coral Gables, Florida.

Dr. Steinman was president of the Dade County Eye, Ear, and Nose Society and the Greater Miami Eye, Ear, Nose and Throat Society. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the Florida Society of Ophthalmology and Otolaryngology, the Southern Medical Association and the American Medical Association. He was also president of the South Florida Hospital Corporation, an organization dedicated to building a new hospital in Miami to adjoin Jackson Memorial Hospital and the medical school.

Dr. Steinman enjoyed music and books at home; he also found relaxation in golf and sailing. He died February 10 of a heart attack. His wife and four children survive him.

HOWARD LEIGH STITT

Howard Leigh Stitt was graduated from the Medical Department of the University of Cincinnati in 1910. He was a specialist certified by the American Board of Otolaryngology. Dr. Stitt practiced in Cincinnati, Ohio, for many years and served on the staffs of the Deaconess, Children's, Good Samaritan, and St. Francis hospitals.

He was an Inactive Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Society of Ophthalmologic and Otolaryngologic Allergy, the American Broncho-Esophagological Association and the American Medical Association. Dr. Stitt died February 15, aged 73, of empyema.

ARTHUR LESLIE STOTTER

Arthur Leslie Stotter, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died September 11, 1957, in Cleveland, Ohio, at the age of 65.

He was graduated from Rush Medical College in 1917, and practiced both ophthalmology and otolaryngology. He was affiliated with Polyclinic, Lakeside and Mount Sinai hospitals in Cleveland. Dr. Stotter was past president of the Cleveland Otolaryngology Club. He is survived by his wife and two daughters.

RUSSELL HOPE STRANGE

Russell Hope Strange was born in Illinois in 1899. He was graduated from Northwestern University Medical School in 1925 and certified a specialist by the American Board of Otolaryngology. He began practicing medicine in Mt. Pleasant, Michigan, in 1931 and was chief of otolaryngology and ophthalmology at Central Michigan Community Hospital there.

Dr. Strange was an Inactive Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Society of Ophthalmologic and Otolaryngologic Allergy and a fellow of the American College of Surgeons and the International College of Surgeons.

His home, Fowler Orchard, gave much pleasure to Dr. Strange. He had a fine stable of riding horses and enjoyed entertaining large groups of friends. He also hunted and fished. Dr. Strange died June 26 and is survived by his wife and three sons.

EDWIN FORBES TAIT

Edwin Forbes Tait was born in Philadelphia, Pennsylvania, August 25, 1894. He attended Brown Preparatory and Temple University of Philadelphia. He received his degree of Doctor of Medicine from the University of Minnesota in 1940, and received a degree of Doctor of Philosophy from the University of Pennsylvania in 1933.

Dr. Tait was a professor of clinical ophthalmology at Temple University Medical School, Philadelphia, and chief of ophthalmology in Montgomery Hospital, Norristown, Pennsylvania. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a fellow of the American College of Surgeons, one of the founders of the National Medical Foundation for Eye Care, vice president of the Medical Society of the State of Pennsylvania, and member of the College of Physicians of Philadelphia, American Society for Research in Ophthalmology, Pennsylvania Academy of Ophthalmology and Otolaryngology, and Pan-American Association for Ophthalmology.

Dr. Tait was an active member of the Central Presbyterian Church of Norristown, Pennsylvania, and was an authority on biblical history. He held the 32nd degree in the Masonic Lodge.

Dr. Tait died in Norristown, September 23. He is survived by his wife, Arita Tait, and two sons, Richard C. and Edwin C. Tait, M.D.

RICARDO TAPIA y FERNANDEZ

Ricardo Tapia y Fernandez was born in Mixquiahuala, State of Hidalgo, Mexico, January 20, 1871. He received his degree of Doctor of Medicine from the Old Faculty of Mexico belonging to the University of Mexico (National) on June 15, 1894.

He was associated with the Hospital San Andres and the General Hospital of Mexico City. Dr. Tapia was founder of the Oto-Rhino-Laryngological Clinic in Mexico, and the founder of the Otolaryngological Section of the National Society of Ophthalmology. He was honorary president of the Mexican Society of Oto-Rhino-Laryngology. He was an emeritus fellow of the National Academy of Medicine and a distinguished member of the Academy since 1912. He was an Honorary President of the First and Fourth Pan-American Congress of Otolaryngology and Broncho-Esophagology.

Dr. Tapia was the founder of several societies in Mexico, including the Red Cross.

He died September 3.

MARTIN CLEVER TAYLOR

Martin Clever Taylor was born in Elliott Boro (Pittsburgh), Pennsylvania, January 10, 1878. He received his degree of Doctor of Medicine from Western Pennsylvania Medical College, Pittsburgh, in 1900. He did postgraduate work in Philadelphia and Montreal.

Dr. Taylor was associated with the Southside Hospital. He was an associate member of the American Medical Association and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Taylor was a member of the Mt. Lebanon Methodist Church. He was affiliated with the Masonic groups.

Dr. Taylor died in Pittsburgh on February 23.

CHARLES ALSTON THIGPEN

Charles Alston Thigpen was born near Greeneville, Alabama, December 19, 1865. He attended Howard College of Marion, Alabama, and received his degree of Doctor of Medicine from Tulane University of Louisiana, New Orleans, in 1888. He did postgraduate work at New Orleans Eye, Ear and Nose Hospital, New York Polyclinic, Royal London Ophthalmic Hospital, and Central London Health Hospital.

Dr. Thigpen was associated with St. Margaret's Hospital. He was a specialist certified by the American Board of Ophthalmology and the American Board of Otolaryngology. He was past president of the Medical Association of the State of Alabama. In 1916, he was a member of the House of Delegates of the American Medical Association. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Laryngological, Rhinological and Otological Society and the Association for Research in Ophthalmology. He was a fellow of the American College of Surgeons.

Dr. Thigpen was a veteran of World War I. He was a successful business man and enjoyed horseback riding as a hobby.

Dr. Thigpen died in Montgomery, Alabama, April 23. He is survived by two daughters, Mrs. Edmond B. Shea and Mrs. Wiley Hill, and one son, Charles A. Thigpen, Jr.

FRANCIS WILLARD THOMAS

Francis Willard Thomas was born in Arcadia, Ohio, April 8, 1889. He received his degree of Doctor of Medicine from

Ohio State University, Columbus, 1912. He did postgraduate work in the Eye and Ear Infirmary of Chicago, Illinois.

Dr. Thomas served on the faculty of the Ohio State University College of Medicine and was on the staffs of Grant and Mount Carmel Hospitals in Columbus. He was a specialist certified by the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Thomas was a member of the St. Albans Episcopal Church of Bexley, Ohio. He was a member of the Masonic Order and the Lions Club. He served in World War I in the Medical Corps.

Dr. Thomas died February 18 in Palm Beach, Florida. He is survived by his wife, Margaret L. and one son, W. M. Thomas, and three granddaughters. One brother and two sisters also survive Dr. Thomas.

HENRY EAGLE THOMASON

Henry Eagle Thomason was born in Cincinnati, Arkansas, April 23, 1877. He received his degree of Doctor of Medicine from St. Louis University School of Medicine in 1902. He did postgraduate work in New York and St. Louis.

He practiced in Kansas City, Missouri, and was associated with St. Mary's Hospital there. He was a member of the Missouri State Medical Association and a Life Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a fellow of the American College of Surgeons. He was also a member of the Jackson County Medical Society.

Dr. Thomason died December 10, 1957, in McAllen, Texas. He is survived by one brother, T. H. Thomason of Summers, Arkansas.

BAXTER HALL TIMBERLAKE

Baxter Hall Timberlake was born December 6, 1900, in Louisburg, North Carolina. He attended Wake Forest College, Wake Forest, North Carolina, and received his degree of Doctor of Medicine from Jefferson Medical School, Philadelphia, 1928. He did postgraduate work at the University of Vienna, Gill Memorial Hospital, Eye, Ear, Nose and Throat Hospital of Roanoke, Virginia, and the Academy of Ophthalmology and Otolaryngology, Chicago, Illinois. He was a specialist certified by the American Board of Otolaryngology.

Dr. Timberlake was associated with Shore Memorial Hospital, Somers Point, the Atlantic City Hospital, Clyde Fish Memorial Hospital and the Children's Seashore House. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a fellow of the International College of Surgeons. He was past president of the Atlantic County Medical Society and served as vice president of the New Jersey Academy of Ophthalmology and Otolaryngology. He was a veteran of World War II.

He was affiliated with the Ventor Community Church. He was a member of the Seaview Country Club and the Atlantic City Country Club.

Dr. Timberlake died in Longport, New Jersey, September 26, 1957. He is survived by his wife, Mary C. Timberlake, and one daughter, Mrs. Peter Walker.

EVERETT HALE TOMB

Everett Hale Tomb was born in 1882. He received his degree of Doctor of Medicine from the University of Pennsylvania School of Medicine, Philadelphia, 1917. He was a specialist certified by the American Board of Otolaryngology in 1930.

Dr. Tomb was associated with the Veterans Administration Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Laryngological, Rhinological and Otological Society. He was a member of the American Medical Association and various local and state medical organizations and a fellow of the American College of Surgeons.

Dr. Tomb died in Boston, October 25, 1957.

GABRIEL FREDERICK TUCKER

Gabriel Frederick Tucker was born November 3, 1880, in Wheeling, West Virginia. He died April 17 in Rosemont, Pennsylvania.

Dr. Tucker was graduated from Jefferson Medical College in 1905, interned from 1905 to 1907 at Mercy Hospital, Pittsburgh, Pennsylvania, and became associated with Dr. B. M. Dickinson until 1910. He moved to Canonsburg, Pennsylvania in 1910 where he engaged in general practice until he entered the army during World War I.

After he was discharged from the army in 1919 he went to Philadelphia, Pennsylvania, where he took special courses in laryngology under Dr. Fielding O. Lewis and in bronchoscopy and esophagoscopy under Dr. Chevalier Jackson. With Dr. Jackson he founded in the Pennsylvania Graduate School of Medicine the first clinic devoted to bronchoscopy and esophagoscopy. When Dr. Jackson left the University of Pennsylvania in 1930, Dr. Tucker was elevated to the rank of professor, and became emeritus professor July 1, 1954. He retired from practice in 1955.

Dr. Tucker was active in teaching bronchoscopy and esophagoscopy and was instrumental in the establishment of departments in that specialty in Misericordia, Bryn Mawr, Abington Memorial and Fitzgerald-Mercy hospitals. He was consultant on the staffs of Mt. Sinai, Germantown and Children's hospitals.

Dr. Tucker was much sought after as a lecturer and his contributions to the literature covered a wide range of discussion on the pathology and treatment of diseases of the chest and larynx. Dr. Tucker was a member of a number of medical societies. He was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1923 and won the Honor Award in 1944.

OSCAR LLOYD VEACH

Oscar Lloyd Veach was born April 14, 1884. He received his degree of Doctor of Medicine from the University of Minnesota. He was a specialist certified by the American Board of Ophthalmology in 1938.

Dr. Veach was associated with Sheridan County Memorial Hospital in Sheridan, Wyoming. He was a member of his county and state medical associations and of the American Medical Association. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a fellow of the International College of Surgeons.

Dr. Veach died November 10, 1957, of aortic aneurysm.

CHARLES BERNARD VOIGT

Charles Bernard Voigt was born in Mattoon, Illinois, October 13, 1874. He received his degree of Doctor of Medicine from the University of Illinois in 1900.

He was the local surgeon for both the Illinois Central Railroad and the New York Central Railroad. He was a member of his county and state medical societies, and was a member of the American Medical Association. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and was a fellow of the International College of Surgeons and the American College of Surgeons. He was associated with the Memorial Hospital.

Dr. Voigt was a Life Elder of the Presbyterian Church. He was the oldest Past Commander of the Godfrey de Bouillon Commandery of Knights Templar of Mattoon and a member of the Masonic bodies. He was a Past Exalted Ruler of Elks; past president of Rotary, and Independent Order of Odd Fellows and Knights of Pythias. He served as director of Okaw Building and Loan Association and director of Central National Bank.

Dr. Voigt died September 8, 1957, in Mattoon. A sister, Mrs. Nellie Freeman of Mattoon, three nieces and one nephew survive him.

HUGH CLAIBOURNE WOLFE

Hugh Claibourne Wolfe was born in Pleasant Garden, North Carolina, December 5, 1892. He received his degree of Doctor of Medicine from the Medical College of Virginia, Richmond, in 1917. Chicago Eye, Ear, Nose and Throat Hospital, Gill Memorial Hospital and Indiana University gave him opportunities for graduate work.

He was on the staffs of St. Leos, Wesley Long Hospital, L. Richardson Memorial Hospital, and Moses H. Cone Memorial Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the American Medical Association. The American Board of Otolaryngology certified him as a specialist.

Dr. Wolfe was a past president of Greensboro Civitan Club and lieutenant governor of the Third District of Civitan International.

Dr. Wolfe died November 9, 1957, in Greensboro, North Carolina. He is survived by his wife, Ann Bagley Wolfe, and one son, Carl Bagley Wolfe, D.D.S.

DORIS LEE SURLES WOOLSEY

Doris Lee Surles Woolsey was born in Dunn, North Carolina, September 27, 1913. She attended Duke University in Durham, North Carolina, and received her degree of Doctor of Medicine from Duke University School of Medicine in 1939. She did postgraduate work at Washington University School of Medicine, interned at Baltimore City Hospital and St. Mary's Hospital in East St. Louis, Illinois, and served a residency at Barnes Hospital, St. Louis, Missouri.

Dr. Woolsey was associated with the Deaconess Hospital, McMillan Hospital, and St. Joseph's Hospital. She was an instructor of clinical otolaryngology at Washington University School of Medicine. Memberships in medical societies include the American Medical Association, the American Academy of Ophthalmology and Otolaryngology and the International College of Surgeons. She was certified by the National Board of Medical Examiners, and a specialist certified by the American Board of Otolaryngology.

Dr. Woolsey was a member of several nonprofessional societies: Altrusa Club; Friends of the City Hospital School of Nursing; and Friends of the City Art Museum.

Dr. Woolsey died February 13, of injuries received in an automobile accident. She is survived by her husband, Robert Dean Woolsey, M.D.; her mother, Mrs. Obie Surles; and one sister, Mrs. Evelyn Bechtold.

JOSEPH WILLIAM WRIGHT, SR.

Joseph William Wright, Sr., was born October 6, 1887. Indiana University School of Medicine granted him a degree in 1911. He was certified a specialist by the American Board of Otolaryngology.

Dr. Wright was on the staffs of St. Vincent's and Methodist hospitals and a consultant in the Indianapolis General Hospital. He was director of Community Hospital. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Laryngological, Rhinological and Otological Society and a fellow of the International College of Surgeons and the American College of Surgeons. He was a member of the American Medical Association and of the Indiana Medical Association, having served as president of the latter in 1952. He helped organize the Indianapolis Society of Ophthalmology and Otolaryngology and served as its first president.

Dr. Wright died October 23, 1957, in Indianapolis. He is survived by his wife and one son, Dr. Joseph William Wright, Jr.

CHARLES AUGUSTUS YOUNG

Charles Augustus Young was born in Baltimore, Maryland, August 20, 1889. He received his degree of Doctor of Medicine from the University of Maryland School of Medicine in 1914. He did postgraduate work in the University of Pennsylvania in the department of ophthalmology.

Dr. Young was on the staffs of the Lewis-Gale Hospital, Jefferson Hospital, Roanoke Memorial Hospital and the Shenandoah Hospital. He was also consultant at the Veterans Administration Hospital in Roanoke. He was a member of various regional medical organizations, of the Medical Society of Virginia, Southern Medical Association, American Medical Association, Association for Research in Ophthalmology, Pan-American Association of Ophthalmology, American Ophthalmological Society, and Roanoke Academy of Medicine. He was a member of and past president of the Virginia Society of Ophthalmology and Otolaryngology and the Wills Eye Hospital Society. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Young was a member of the Christ Lutheran Church of Roanoke. He was interested in traveling, books, woodworking, and the United States Navy. He served in the Medical Corps of the United States Army during the first World War and in the Medical Corps of the United States Navy during the second World War. He was a member of the Military Order of the World Wars. He also held membership in the Masonic Order, the Roanoke Country Club, Phi Sigma Kappa and Phi Chi.

His awards included the Victory Medal with three stars during World War I; and the Victory Medal, the American Defense Service Medal, the American Area Campaign Medal, and the U. S. N. R. Medal during the second World War.

He was appointed a member of the State Board of Opticians in 1954.

Dr. Young died July 25, 1957, in Roanoke, Virginia. He is survived by his wife, Mrs. Jettie C. Young, one son, Dr. Charles A. Young, Jr., and two daughters, Mrs. Lynwood W. Richardson and Miss Helen E. Young.

WILLIAM ZENTMAYER

William Zentmayer was born in Philadelphia, October 28, 1864. He received his degree of Doctor of Medicine from the University of Pennsylvania in 1886.

Dr. Zentmayer was a consulting surgeon at the Wills Eye Hospital and an emeritus professor of ophthalmology at University of Pennsylvania Graduate School of Medicine, Philadelphia.

He was an associate member of the American Medical Association, and was chairman of its Section on Ophthalmology in 1916-1917. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and past president of the American Ophthalmological Society and the Association for Research in Ophthalmology.

He was awarded the Howe Medal by the American Ophthalmological Society in 1948. In 1945, the St. Louis Society for the Blind presented the Leslie Dana Gold Medal to Dr. Zentmayer. He was a director of the National Society for Prevention of Blindness and a member of the Ophthalmological Society of the United Kingdom.

Poetry and music were important to Dr. Zentmayer and he valued his membership in the Pennsylvania Academy of Fine Arts. He also was a member of the Philadelphia Academy of Natural Sciences. He was on the editorial board of the A.M.A. Archives of Ophthalmology. Dr. Zentmayer died March 18 in Merion Station, Pennsylvania. He is survived by one sister, Mary Zentmayer.

BOOK REVIEWS

SYSTEMIC OPHTHALMOLOGY. Edited by Arnold Sorsby. Second Edition. London, England, Butterworth; and St. Louis, C. V. Mosby; 1958. 666 pp.; 277 figures and illustrations, many in color; bibliography and index. $25.00.

The first edition of Sorsby's *Systemic Ophthalmology*, published in 1951, was designed to cover more than the medical aspects of eye disease as they were treated in books written on medical ophthalmology, "for the general aspects of eye disease carry surgical, obstetric, metabolic, dermatologic and other implications no less than those of a purely medical character." (Preface to the First Edition.) After seven years a second edition has made it possible to bring the book up to date and to delete unimportant matter. This book is unlike the usual textbook in that it is a volume of contributions on selected subjects by collaborators chosen for their special interest in and knowledge of systemic diseases by category. There is a minimum of overlapping by the authors without omission of important material where the distinction of disease by diagnosis allows application of general terms. Of the thirty-four collaborators, ten are American doctors, others are English or English trained and are well known through previous writing.

The book is divided into six parts; several writers contribute to more than one part. Part 1 is on Prenatal Pathogenic Influences; Part 2, Inflammation, Allergic Reactions and Infections; Part 3, Nutritional, Metabolic and Endocrine Disturbances; Part 4, Central Nervous System; Part 5, Cardiovascular Systems; Part 6, Other General Disturbances. As the title implies, there is nothing in the book on refraction, surgical techniques, anatomy or physiology. There is a wealth of material here that is also available in ophthalmic journals of the past ten years, but arranged especially for this volume it is complete, orderly and authoritative. Not a teaching textbook for residents, it is a must for consulting ophthalmologists and invaluable for internists, neurologists, allergists, dermatologists and general physicians.

W.L.B.

APPLIED PHYSIOLOGY OF THE EYE. By H. Willoughby Lyle, M.D., assisted by T. Keith Lyle, M.D., Baltimore, Williams & Wilkins, 1958. Cloth; 298 pp.; 32 line drawings. $9.00.

Physiology of the eye encompasses the central nervous system and morbid conditions of other organs. To appreciate fully the scope of the field, a brief anatomic description of the eye and adnexa is required before relating the functions of the parts and their interrelationship. Consequently the authors have taken each of the structures; eyelids, eyeball, pupils, uveal tract, lens, vitreous, retina, optic nerve and some neurologic centers, and have combined a brief description of the anatomy with a delineation of symptoms of disorder and normal function.

In many of the twenty-three chapters are clinical descriptions of pathologic conditions of the eye that appear in all textbooks and serve as background for the comments on deviations from the normal. In many instances the comments on a particular item may be reduced to a definition. While the book contains a great deal of valuable information, the subject matter is so condensed and briefed that it amounts to a well kept notebook. This is what the

authors evidently intended. As such it is particularly helpful to students and candidates for certification by examining boards, to practitioners for review and to teachers as a handy guide. It is recommended for first year residents in ophthalmology and students in allied disciplines.

W.L.B.

AUTHOR-PUBLISHER-PRINTER COMPLEX. By R. S. Gill. Third Edition. Baltimore, Williams & Wilkins, 1958. Cloth; 131 pp.; 9 figures. $2.25.

The third edition of this discourse on the art of publishing scientific papers and books is a comprehensive outline of the steps that must be taken to produce the final form of publication. The purpose of the book is to tell the author what he needs to know in order to make the best use of his talents and the time required by the editor, the redactor and the printer to make possible publication in the best form. In eleven short chapters the subjects cover the duties of the author, typist, proofreader, illustrator, publisher and bookseller. Punctuation, phraseology, chapter headings, subheadings, type style and size, and a dozen other details must be left to the redactor and publisher if the author fails to furnish the manuscript with instructions.

Everyone who writes a scientific paper for publication in a journal or as a chapter in a book could very well start his task with three essentials: paper, typewriter, and a copy of this book. He will save more than enough on each paper to pay for the book. If the information given therein is heeded, the author will gain the esteem of the people who make the final production possible and the paper will be his own and not an artificially polished set of notes. I speak with some feeling on the matter.

W.L.B.

SCIENTIFIC AND TECHNICAL PAPERS. By Sam F. Trelease. Baltimore, Williams & Wilkins, 1958. Cloth; 176 pp.; No illustrations; index. $3.25.

From choosing a research problem to final proofreading the author presents the way in which a scientific paper should be prepared. The way to write medical and other scientific papers has occupied the attention of editors and publishers particularly, but also of readers, for prophylaxis is preferable to therapy when comparable results can be obtained. Proper tabulating, indexing, illustrating, composing and expressing are quite important features in every case of publication and trained expert redactors must sift through pages of manuscript just to tell the printer what to do. Often the redactor must go back to the author with a question that requires rewriting to clear up ambiguity or a suggestion as to rearrangement of text, figures and tables.

Only the editorial department of a publishing establishment realizes the mass of detail that goes into the manuscript of every paper presented for publication. Some editorial policies are based on individual preferences of the editor. These need not concern the author, but such things as good form and usage, footnotes, references, tables, drawings, graphs, illustrations and photographs are standard with all publishers and should be understood by the author before he gets his manuscript underway. This little book is replete with just the information the author wants on technical writing. It also has pages on library use and statistical methods. Even experienced writers who consult this book before starting their manuscript will save hours of rewriting and polishing.

W.L.B.

VETERINARY OPHTHALMOLOGY. By R. H. Smythe, M.R.C.V.S. Second Edition. Baltimore, Williams & Wilkins, 1958. Cloth; 59 figures; 16 plates, photographs and photomicrographs; index. $8.50.

A great deal has been written on comparative anatomy of the eye and on the structure, position and function of the eyes of animals. The advancement in veterinary science in almost every other direction made necessary a compilation of knowledge concerning the disorders of animal eyes that may be applied to the treatment of animals.

The work is divided into two parts: Anatomy and Physiology of the Eye and Clinical Ophthalmology.

The second part distinguishes this book from the treatises on the eyes of animals, birds and reptiles that fill many textbooks. The eyes of domestic animals suffer injuries, burns and diseases similar to those found in human eyes. Treatment, however, is another matter. Under *Sympathetic Ophthalmia* is found the following: "Affection of a hitherto sound eye 'in sympathy' with its fellow eye is of very infrequent occurrence in animals other than man." "Infection of the second eye, in our patients, is the result of generalized infection and not the so-called sympathetic ophthalmia, so well known in man." Many inflammatory conditions improve spontaneously in the course of time, and those which do not behave in this way are seldom helped by treatment. Several pages are devoted to treatment, formulas, prescriptions and therapeutic procedures.

W.L.B.

CLEFT PALATE. By Muriel E. Morley, M.Sc. Fourth Edition. Baltimore, Williams & Wilkins, 1958. 260 pp. $6.50.

This book is written for speech therapists. The author presents anatomic descriptions of the oral cavity, particularly clefts of the lip and palate, through the embryonic and infantile stages of development and traces the functional impairment that results from abnormal development of these parts. The improvement in surgical repair of cleft palate and harelip has resulted in better speech accomplishments by experienced therapists and improvement in orofacial appearance and function. There is a comprehensive bibliography but few footnote references. It is worthwhile reading for the oral surgeon.

W.L.B.

News - - - Notes

THE FIRST AFRO-ASIAN CONGRESS OF OPHTHALMOLOGY

Dear Sir,

The subject of trachoma was one of the main subjects of study in the first Afro-Asian congress of ophthalmology, held in Cairo from 1-5 March, 1958. The question of trachoma stage 4 was discussed and it was unanimously decided that this stage of trachoma (T4) should be omitted from all classifications of trachoma. Some of the reasons for this decision are:

1. It is universally agreed that when trachoma heals completely, the condition is called trachoma 4. So trachoma 4 is a condition in which the patient is completely cured, is noninfectious and has no trachoma any more, and it is completely ridiculous to consider such a condition as one of the stages of the disease. A similar state of affairs is not known in medicine. Thus a syphylitic patient who is cured is not called syphylitic stage 4, and a tuberculosis patient who is cured is never stigmatised as tuberculosis stage 4. This in spite of the fact that in both diseases, scars, analogous to the scars of trachoma 4, may be present in the cured patient.

2. Trachoma 4 is a great source of inexactitude and confusion as far as statistics, local or international, are concerned. Thus, and just as an example, if we examine the population of one of the countries where trachoma is prevalent; and classify the cases according to one of the classifications that consider trachoma 4 of the stages of the disease, we may find the percentage of trachoma in that country 60 per cent, for example. Out of these cases, 40 per cent may be trachoma 4, 10 per cent trachoma 3 and 10 per cent trachoma 2 and trachoma 1. Thus, in reality, the percentage of the incidence of trachoma in that country is only 20 per cent and not 60 per cent and the percentage of cases that need treatment is 20 per cent and not 60 per cent. So the classifications that consider trachoma 4 as one of the stages of the disease are inexact and give a false information regarding the incidence of the disease. This has a great bearing from the scientific, epidemiologic, economic and other points of view.

3. If a cicatrised lid should be given a name let it be called a "cicatrised lid." Otherwise what is the benefit of calling such a condition trachoma 4. As far as we know, there is no benefit of such a nomenclature, while its great disadvantages are many.

For all these reasons, the first Afro-Asian congress of ophthalmology, comprising members from the following countries: Ceylon, China, India, Indonesia, Iraq, Japan, Jordan, Lebanon, Philippine, Saudi Arabia, Soudan, Thailand, Tunis, Turkey, U.S.S.R. and The United Arab Republic (Egypt and Syria) took the following resolution:

It is decided that trachoma stage 4 should be omitted from all classifications of trachoma. The decision is to be declared to all concerned international organisations.

The decision will be recommended to be put in action by member countries.

Yours Truly,
SABRI KAMEL, M.D.
General Secretary

Dar-el-Hekma, 42 Kasr-el-Ainy Street,
Cairo, 15 June 1958.

SCHOENBERG LECTURE

Dr. Harold G. Scheie, professor of ophthalmology at the University of Pennsylvania, will give the annual Schoenberg Memorial Lecture 8:15 p.m., December 1, at the New York Academy of Medicine, 2 E. 103rd St. He will discuss "Management of Infantile Glaucoma."

The Lecture is sponsored jointly by the New York Society for Clinical Ophthalmology and the National Society for the Prevention of Blindness to honor Dr. Mark J. Schoenberg. Before his death, in 1945, he was prominent in the work of both societies.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

The Association for Research in Ophthalmology, Inc., will publish a second Supplement to contain papers presented at the Section meetings. Any author desiring publication in this Supplement should send his completed manuscript and any discussion to Lorand V. Johnson, M.D., 10515 Carnegie Ave., Cleveland 6, as soon as possible. The absolute deadline is February 1. The Supplement will be published before the June national meeting.

Letters of intention to present papers at the June meeting should be sent to Dr. Johnson immediately. All abstracts must be received by December 15.

CALL FOR EXHIBITS

The 1959 meeting of the Section on Ophthalmology of the American Medical Association will be held in Atlantic City, June 8 through 12, simultaneously with that of the Association for Research in Ophthalmology.

A $250 prize is offered for the best scientific exhibit prepared for the Section at each Scientific Session. Anyone wishing to prepare a scientific exhibit should communicate with Frank W. Newell, M.D., Representative to Scientific Exhibit, 950 E. 59th St., Chicago 37, as soon as possible.

POSTGRADUATE COURSES

The Department of Laryngology and Broncho-Esophagology, Temple University Medical Center, is offering two postgraduate courses. A course in Bronchoesophagology will be offered February 16 to 27 and a course in Laryngology and Laryngeal Surgery will be offered April 13 to 24. Drs. Chevalier L. Jackson and Charles M. Norris are arranging the courses.

The tuition fee for each course is $250. Application blanks and further information concerning dates of subsequent courses may be obtained by writing Jackson Research, Lab 604, Temple University Medical School, 3400 N. Broad St., Philadelphia 40.

RESEARCH STUDY CLUB OF LOS ANGELES

The Research Study Club of Los Angeles will hold its Twenty-eighth Annual Midwinter Convention in Ophthalmology and Otolaryngology, January 26 through 30, at the Ambassador Hotel, Los Angeles.

Drs. A. Franceschetti, Geneva, Arthur G. DeVoe, New York, Lester T. Jones, Portland, Ore., and Arthur Jampolsky, San Francisco, are guest speakers in ophthalmology. Drs. Franceschetti, Jones, Jampolsky and Carrol L. Weeks, Los Angeles, will give instruction courses in ophthalmology.

Guest speakers in otolaryngology will be Drs. Francis L. Lederer, Chicago, F. Johnson Putney, Philadelphia, and Victor Goodhill, Howard P. House, Max Pohlman and Joel J. Pressman, all of Los Angeles. Drs. Lederer, Putney and W. E. Compere, Jr., Manuel R. Wexler and E. W. Johnson, Ph.D., all of Los Angeles, will give instruction courses.

A special course in cardiac resuscitation will be given by Dr. William P. Mikkelsen of the University of Southern California. This Course, including laboratory demonstration, will be available to the first 25 applicants for an additional fee of $50.

The fee for the convention, including all Round Table Luncheons, is $110 and should be sent to Norman Jesberg, M.D., Treasurer, 500 S. Lucas Ave., Los Angeles 17.

FLORIDA MEETING

The Thirteenth Annual Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held February 16 through 21 at the new Americana, 9701 Collins Ave., Miami Beach.

Lectures on otolaryngology will be delivered February 16 through 18 by Drs. John J. Conley, New York, George T. Harrell, Gainesville, Fla., William C. Huffman, Iowa City, P. E. Ireland, Toronto, and Arthur L. Juers, Louisville, Ky.

The ophthalmologists to lecture February 19 through 21 are Drs. Frank D. Carroll, New York, Paul A. Chandler, Boston, John W. Henderson, Ann Arbor, Mich., and Harvey E. Thorpe, Pittsburgh.

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

Dr. George A. Sisson, Syracuse, N. Y., will be the guest speaker at the regular fall meeting of the New England Oto-Laryngological Society. The Society will meet November 19 at the Massachusetts Eye and Ear Infirmary, Boston.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

Dr. Irving B. Goldman's discussion of "Correction of a Deviated Nose" and a colored motion picture with sound on "Elective Rhinoplasty" will highlight the Scientific Session of the meeting of the American Society of Facial Plastic Surgery, November 12. The Society will meet with the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings at the Academy of Medicine of Brooklyn, 1313 Bedford Ave., Brooklyn.

SOUTH AFRICAN VISIT

Dr. Ralph W. Ryan, Morgantown, W. Va., traveled to the Union of South Africa to present a paper before the Conference on Industrial Ophthalmology held in Johannesburg, November 10 through 12. The Conference met to set up a program of industrial eye safety measures for the rapidly growing industries of that country.

Dr. Ryan also presented a paper before the annual meeting of the South African Ophthalmological Society in Johannesburg on "Toxoplasmosis as Related to Eye Disease."

POSITIONS AVAILABLE

An unusual opportunity for an OTOLARYNGOLOGIST is available on the Pacific Coast. This town of 350,000 offers an excellent chance for private practice. If interested, write Joseph Fagin, M.D., Medical Center, Los Altos, 3115 E. Ocean Blvd., Long Beach 3, Calif.

An OTOLARYNGOLOGIST is needed in Claremont, California. Write Morrill L. Illsley, M.D., P.O. Box 24, Claremont, Calif.

An OTOLARYNGOLOGIST is wanted to be head of the department in a 150-bed county hospital in a city of 22,000. There is also an opportunity for private practice. For further information write Mr. W. B. Barnhart, Hospital Administration, Columbia, Tenn.

A physician who is returning to teaching medicine wants an OTOLARYNGOLOGIST to take over his practice in a growing Midwest city with 250,000 population trade area. The practice, established for 38 years, yields an unopposed $50,000 a year. It is associated with two Board-certified ophthalmologists. If interested contact J. B. Gregg, M.D., 1600 S. Western Ave., Sioux Falls, S. Dak.

Listings of positions available should be sent to W. L. Benedict, M.D., 15 Second Street S. W., Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist: diplomate, resident, etc.) (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

FOREIGN SUBSCRIPTIONS

Some foreign members of the Academy as well as subscribers to the TRANSACTIONS are experiencing difficulty in remitting in American dollars. For their convenience the Academy will accept any legal foreign currency in payment of dues, fees for Home Study Courses, Teachers' Manuals, Orthoptic Journals, TRANSACTIONS, etc., at the prevailing rate of currency exchange.

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